HYPERTENSIVE DISEASES

Causes and Control

By

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TO

JANET

who made this book come into being

It is the obligation of those who have learned to teach

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PREFACE

ne to watch the gradual unfolding of
Before therapy becomes a reality
are gathered and minor and major
theories are evolved tested and discarded Step by step advances are then made and small disconnected islands of tested facts slowly appear in the chaotic morass of the unknown Finally a theory embracing and consistent with these facts evolves resilient enough to include new ones (therefore like prices subject to change without notice) but resistant enough to exclude unsupported fancies and theories Thus the direction for new experiments is made clear and some of the blank and unknown area charted

Long before a majority of the processes are known which in their derangement lead to disease the temptation arises to test the theory by attempting reversal of some of them To yield to that temptation is justifiable only when the result is new information and the further exploration of a derangement For reversal of a disturbance of the human organism toward normal is fraught with many danger until enough is understood of underlying processes and of the whole organism to allow a logical and confident approach Yet that time becomes ripe at a certain point of progress and when it does therapy naturally evolves therapy directed not only at one or more disturbances but principally at disturbances f

processes and apply that understanding to the basic disturbances which we call disease One must speculate and intuitively experiment with fundamental changes in metabolism which are chemical and in stresses which are physico-chemical One must constantly strive to simplify the apparently complex to break down into its constituent parts that which appears vague and disjointed In this there comes the reward of understanding for Nature operates under simple basic laws tested over time Only in the imitation of Nature can man progress to knowledge and in the field of Man

until the framework of the small amount of knowledge available Thus they can be confined into compartments labeled not understood and shelved for future study Those which are capable of exploration and which offer the most promising ap

Read not to contradict and confute, nor to believe and take for granted, nor to find talk and discourse, but to weigh and consider.

—SIR FRANCIS BACON

The highest happiness of man as a thinking being is to have probed what is knowable and quietly to revere what is unknowable.

—GOETHE

Something hidden. Go and find it. Go
and look behind the Ranges—
Something lost behind the Ranges. Lost and
waiting for you. Go!

KIPLING

Contents

system kidneys adrenals, and possibly liver Because maintenance of blood flow is as important to the body's economy as is maintenance of oxygen tension we are dealing with very fundamental cellular functions

local or general circulatory insufficiency such as peripheral vascular collapse from trauma or hemorrhage congestive circulatory failure local arterial diseases causing ischemia and the like Arterial hypertension is therefore not to be considered the result of a disturbance unique in itself bu

theories

up lamental

difference in the smooth muscle of arteriolar walls of individuals with hypertension an hereditary or congenital defect the manifestation of which is a ensitivity greater than normal to the action of normal amounts of circulating or locally manufactured vasoconstrictor substances The other presupposes normal vascular reactivity to increased amounts of vasoconstrictor substances The present discussion is concerned primarily with the latter

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Pathogenic Influences.—Of these there are at least three (1) a *neurogenic* factor, action of which results in vasospasm (2) a nephrogenic factor, which results from renal ischemia induced either by vasospasm organic renal vascular or parenchymal diseases or both (3) an adrenocorticogenic factor, which may result from psychic stress in certain individuals constituted in such a way that adrenal discharges are common

The effects of hypertension are two fold at least (1) the results of continued physical strain on heart, blood vessels and kidneys and (2) metabolic abnormalities which result from restricted blood flow in special organs In addition the unfortunate characteristic of sustained intra-arterial tension to hasten the development of atherosclerosis is considered one of the effects

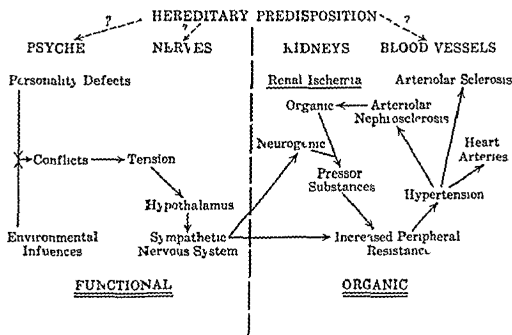


Fig 1 —Theoretical pathogenesis of arterial hypertension [1-3] The hereditary predisposition may be manifest by changes in the psyche autonomic nerves or blood vessels However it acts there are certain disturbances of personality which in a complex environment lead to conflicts The emotional tension resulting therefrom produces autonomic discharges by way of the hypothalamus The result is neurogenic vasospasm When vasospasm involves the kidney humoral vasoconstrictor substances are released Repetition of the process eventually leads to the vicious circle of renal arteriolar disease organic renal ischemia more pressor substances and so on upon which the neurogenic influence is superimposed Removal of the neurogenic influence would not result in reversal of the process unless organic renal ischemia were absent At the left is pictured the functional or psychoneurogenic component and at the right the organic or somatic component

The condition itself is therefore the result of many interacting influences and factors each one influencing the other and each dependent upon the other, many organs are involved, notably the brain autonomic nervous

VISCOSITY OF BLOOD

The second formula is one which appears more complicated It was developed by Poiseuille on principles of fluid dynamics Unfortunately it does not explain adequately the behavior of blood a fluid containing almost half its volume in the form of small distortable particles (red blood cells) flowing through tubes of decreasing size It is adequate as an approximation and quite exact for water or a homogeneous fluid

$$\text{Pressure} = \frac{8 \times \text{Length of Tubes} \times \text{Viscosity} \times \text{Flow}}{\pi \times \text{Radii of Vessels}^4}$$

Mathematically minded readers who have the perseverance to track down complicated theory are referred to the publications of Lamport[1] and Green who have thrown some light on this subject It means that peripheral resistance varies directly with the viscosity and inversely with the fourth power of the sum of the radii of the arterioles Although viscosity is believed to be normal in hypertension the following extreme examples indicate the manner in which changes of viscosity can affect blood pressure

	Mean B P mm Hg	*Cardiac Output L min*	*Peripheral Resistance (1000 units)*	*Viscosity (poises)*	*Vascular Bed*	*Calculated B P without Peripheral Compensation mm Hg*
Normotension	100	4	25	035	normal state	100
Polycythemia	128	4	32	045	normal state	128
Polycythemia	150	6	25	045	vasodilatation	113
Polycythemia	180	4	45	045	vasoconstriction	194
Anemia	100	4	25	015	vasoconstriction	43
Anemia	100	8	12 5	015	vasoconstriction	86
Anemia	100	3 3	10 [illegible]	015	normal state	100
Anemia	80	10	5	015	vasodilatation	171

TABLE 1.—ELEVATED BLOOD PRESSURE CAUSED BY PRIMARY FACTORS OTHER THAN GENERALIZED VASOSPASM

Condition	*Systolic pressure*	*Diastolic pressure*	*Stroke volume*	*Effective cardiac output*	*Blood volume*	*Blood viscosity*	*State of arteriolar bed*	*Remarks*
Cardiac Factors								
Complete Heart Block	High	Low	High	N	N?	N	N or dilated	Arteriolar run-off greater
Aortic Insufficiency	High	Low	High	N or low	N	N	Dilated	Arteriolar run-off normal
Marked Bradycardia	High or N	Low	High	N	N	N	N	Arteriolar run-off normal
Peripheral Factors								
Arteriovenous Aneurysms	High	N or low	High	High	Increased	N	N or constricted	Resistance locally low
Atherosclerosis	High	N or low	N	N	N	N	N or dilated	Arterial elasticity reduced, vessels rigid
Cardiac and Peripheral Factors								
Patent Ductus Arteriosus	High	N or low	High	N	Increased	N	N or constricted	Resistance locally low
Hyperthyroidism	High	N or low	High	High	N or increased	N	Dilated	Blood flow rapid
Hemic Factors								
Polycythemia	High	High	N or low	N or low?	Increased	Increased	N?	Resistance high due to viscosity
Increased Blood Volume	Slightly high	Slightly high	High	High	Increased	N	N	Increased cardiac output and/or venous back pressure
True Arterial Hypertension	High	High	N	N	N	N	Constricted	(for comparison)

N = Normal state

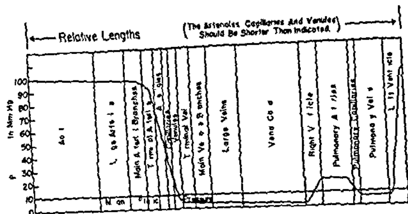


FIG 4 —The fall of pressure in the circulation (i e resistance) through various anatomical divisions of the vascular system (after Green) The relative length of each

Therefore the total resistance equals the sum of the reciprocals of the resistances of each circuit or the sum of the conductances of each circuit resistance being the reciprocal of conductance

$$\frac{1}{\text{Total Resistance}} = \frac{1}{\text{Resistance of Brain}} + \frac{1}{\text{Resistance of Kidneys}} + \frac{1}{\text{Resistance of Splanchnic Bed}} + \frac{1}{\text{Resistance of Muscles}} + \frac{1}{\text{Resistance of Bone}}$$

etc * It is obvious that if the resistance of only one area were increased blood would be shunted into other areas of unchanged resistance (increasing the flow) and relative ischemia of that area would occur On the other hand if the resistances of all areas were increased equally and proportionally to the blood flow through each area total resistance would be increased without redistribution of blood from one area to another Actually blood is being constantly redistributed in response to the metabolic needs of each area much more going to muscles during exercise than at

The same holds true for electricity ... to give the total for a circuit with a ... Total Resistance = R + R + R + R ... total equals the sum of their reciprocals

$$\frac{1}{\text{Total Resistance}} = \frac{1}{R} + \frac{1}{R} + \frac{1}{R} + \frac{1}{R}$$

with more current ... drop other ... that ...

VASOMOTOR TONE

The size of the arteriolar bed is of paramount importance to the problem of circulatory hemodynamics,* but, unfortunately, there are no direct method for measuring it. Recently two have given some promise: *critical closing pressure*[3] which is the pressure at which flow ceases in a local vascular bed after arterial occlusion, and the end pressure of the systolic arterial pressure gradient[4] in a local circulation. Both methods can be used in anesthetized animals, but do not apply to clinical studies. An adaptation of the second can be used in man's forearm or leg only, by occluding arterial flow with a cuff. Valuable information has resulted from the use of one of these methods in *anesthetized* hypertensive dogs: the most valid function of the level of blood pressure was found to be the size (cross-sectional area) of the splanchnic vascular bed.

A very small change in the diameter of every arteriole in the body will alter blood pressure markedly, other factors remaining constant. A rough approximation of the per cent of constriction has been made which, although subject to wide errors, is revealing. The calculation assumes that 100 per cent constriction means all vessels are closed tight.

Per cent of total Constriction		*Mean B.P. mm. Hg*	*Cardiac Output L/min*	*Peripheral Resistance (1000 units)*
Normotension	70	100	4	25
Hypertension	77	150	4	37.5
Hypertension	79	200	4	50
Hypotension	60	80	4	20

Therefore it can be seen that very small degrees of vasoconstriction can cause profound alterations in the level of blood pressure. It may be that under some circumstances resistance can vary as the fifth power of the radius of the vessels. The relative resistances of the vessels are shown in Figure 4.

DISTRIBUTION OF PERIPHERAL RESISTANCE

The circulation through various organs and tissues is arranged in parallel, a large number of parallel circuits being fed directly from the aorta and its larger branches. Parallel circuits also exist within each organ (Fig. 5).

* According to Green[2] the average drops in pressure along the various sized arteries of a dog are as follows (mm. Hg): Aorta and large arteries 2.8, main branches 4.6, secondary branches 4.8, tertiary branches 13.4, terminal arteries 4.5, terminal branches 4.0, arterioles 8.6. Therefore it appears that the pre-arteriolar small arteries contribute to a greater proportion of the resistance than do the arterioles. The cross-sectional areas of the tertiary branches are 11.7 sq. cm., of the terminal arteries 19.6 sq. cm., of the terminal branches 91 sq. cm., and 125 sq. cm. for the arterioles. If this is so, one can consider hypertension the result of spasm of both small arteries and arterioles of all sizes. Green's contributions are well worth study.

pressure against which it is pumped Both an increase in output and an increase in pressure will increase the amount of work performed by the heart * The result is hypertrophy of the muscle from overwork Second

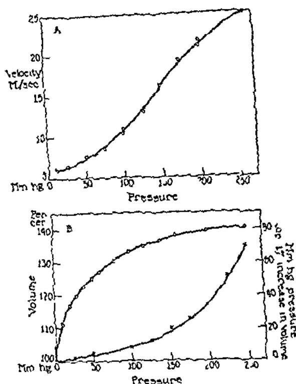


Fig 6 A The relation of pressure to velocity of the pulse wave in the normal brachial artery ... taken from the arm of a man aged forty years eighteen hours after death The curve would be steeper if the artery were sclerotic B Upper curve The progressively declining increase in arterial volume with each successive increase in pressure As the pressure rises the arterial volume approaches the elastic limits of the artery until further increase of pressure can ...

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the volume of blood in the arterial circulation is increased Arterial volume is a function of pressure within the limits of elasticity (Fig 6) The arterial walls are stretched by the increased pressure (and volume) the result is a more rapid transmission of the pulse wave along less elastic tubes The effect is to all intents a physiological hardening of the arteries due to their lessened elastic rebound Probably their smooth muscle is stimulated by the pressure or by circulating chemical substances

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TABLE 2 – RESTING DISTRIBUTION OF BLOOD IN THE VASCULAR BED AND REGIONAL BLOOD FLOW AND OXYGEN USAGE (AFTER BAZETT*)

Blood distribution	ml		ml
Heart	250	Aorta	100
Pulmonary arteries	400	Systemic arteries	150
Pulmonary capillaries	60	Systemic capillaries	300
Saccular venule	140	Venules	200
Pulmonary vein	700	Systemic vein	2050
Total Pulmonary System	1300	Total Systemic Vessel	3100
and Heart	250		
	1550	Unaccounted 550 (probably extra blood in reservoirs of liver and spleen)	

Blood flow	Weight of tissue Kg	O_2 Use ml/min	O_2 Uses ml/Kg/min	Blood flow ml/sec	Blood flow ml/Kg/sec	Resistance R units	Arterio-venous O_2 difference	Remarks
Portal System	2.6	51	19.6	25.0	9.6	3.6	3.4	High flow
Kidney	0.3	14	46.7	21.0	70.0	4.5	1.1	High flow low resistance
Central Nervous System	1.4	46	32.8	12.5	9.1	7.2	6.2	High O_2 consumption
Muscles	31.0	50	1.6	14.0	0.5	6.4	6.0	Flow low at rest
Skin	3.6	12	3.3	7.0	1.9	11.7	3.0	
Heart Muscle	0.3	27	90.0	3.3	11.0	27.0	17.5	High O_2 consumption
Other Organs Skeleton etc	23.7	50	2.1	13.5	0.6	6.7	6.2	
Total	63	250	4.0	97	1.5	0.93*	4.3	

* Assuming a mean pressure of 90 mm Hg. The total is the sum of the reciprocals.

Note. The above figures were calculated for a thirty year-old man weighing 63 Kg. Height 178 cm. Blood volume 5.2 L. Mean arterial pressure 90 mm Hg. Cardiac output 5.8 L/min. On this and other subjects Dr Bazett's thinking was profound and his understanding outstanding*

Chapter

3

DEFINITIONS

THE definitions given in the following discussion are for the most part those which have been commonly accepted.[1] Minor modifications have been made in an attempt to denote the degree of general involvement and advance of the condition. Serious secondary pathological accidents (coronary occlusion, cerebral hemorrhage, renal insufficiency) are not considered in the stages of progress of the condition, for they are usually difficult to predict and represent secondary effects, although most important ones to the sufferer. The various types of hypertension, judged from etiological and pathogenetic viewpoints, will be defined in appropriate chapters.

ARTERIAL HYPERTENSION

Arterial hypertension is merely a physical finding, like fever, elevated

system. The underlying diseases may involve the parenchyma of the kidneys or their blood vessels, the nervous system, the endocrine glands, especially the pituitary-adrenal axis, or more rarely, the volume and viscosity of the circulating blood, or the output of the heart. Transient hypertension may be a functional alteration of one or another of the factors which contribute to circulatory homeostasis.

Arterial hypertension is defined as a condition in which the peripheral resistance to the flow of circulating blood is chronically increased to such a point that the diastolic blood pressure is 90 mm. Hg or more as determined by the indirect method using the disappearance of sound as a criterion. When this is so, the systolic pressure is usually 140 mm. Hg or more. A definition such as this one does not explain the true state of

under many severe conditions may be in marked vasospasm without elevation of blood pressure (hemorrhage, shock, cardiac damage, severe anem... The term *generalized vasospasm* is a more inclusive one; arterial hypertension is a manifestation of generalized vasospasm when cardiac o...

to contract somewhat Since most of the increased resistance to flow is caused by arteriolar constriction, there is apparently little effect upon capillaries or tissues, although of that we are not certain Thirdly, there may be a change in the amount of blood at any one time in different arterial systems, the predominantly elastic aorta containing more and the predominantly muscular arteries containing normal amounts, or even less Certainly the arteries of the retina appear as if their calibers were decreased The pathological and chemical changes will be discussed at length later

Factors Necessary for the Maintenance of Hypertension —Some clinicians and even investigators, lose sight of the fact that integrity and proper function of most or all of the organs and systems which contribute to the maintenance of blood flow and blood pressure are necessary in order that hypertension (or normotension) can exist Unfortunate therapeutic attempts have been, and will be made, when this is not considered Naturally the heart must function adequately, normotension induced by acute coronary occlusion is not a healthy state *Blood volume must be adequate* to maintain cardiac output Blood viscosity must be adequate to provide resistance to flow through small vessels The adrenal cortex must function properly Addison's disease and hypertension are inconsistent states unless the former is treated The liver must have adequate function and the lungs oxygenate the blood Large arteriovenous fistula must not be present total peripheral resistance will fall unphysiologically at the expense of vital tissues The autonomic nervous system must usually be present although this is not always true in the experimental animal and nervous centers governing vasomotor controls active Extensive tissue destruction or serious inflammatory or pyrexic disease must be absent These and probably other essential functions are necessary for hypertension to exist and methods of treatment based on disturbing them to alter abnormal numbers may be unphysiological illogical and unhealthy

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TABLE — STAGES IN THE COURSE OF ARTERIAL HYPERTENSION

Stage	Blood pressure	Approximate diastolic mm Hg	Degree of arteriolar disease (Hypertensive vascular [illegible])	Ocular fundi grade	Rate of progress	Vascular compensation	Remarks
0 Prehypertension	Usually normal. Slightly elevated under stress	85–95	0	0	Variable	Complete	No symptoms. No damage unless progress to other stages
I Mild Benign Hypertension	Normal at night and at rest. Elevated by day and in activity	90–105	0*	0–I	Variable	Complete	Consistent with long life. May revert to normal
II Moderate Benign Hypertension	Fluctuating. Normal only under heavy sedation	105–120	0 +	I–II	Slow	Complete unless [illegible]	Consistent with long life unless complicated. Occasionally reverts to normal
III Severe Benign Hypertension	High fluctuating, never normal	120–160	+ ++++	II	Usually rapid (years)	Partial to decompensated	Apoplexy or heart failure may develop. Long life rare
IV Malignant Hypertension a) Early	High, fairly fixed. May fall with sedation	120–160	+ ++++	III	Usually rapid but reversible	Partly decompensated	Danger signal for downhill course
b) Severe	High fixed. May fall with sedation	130–200	++ ++++	IV	Rapid (months)	Partly decompensated	Usually fatal unless interrupted
c) Decompensated	Variable, usually high	130–200	++++	IV	Rapid (weeks)	Decompensated (N retention)	Renal insufficiency and heart failure. Always fatal

Note. The course can be modified at any stage by the development of [illegible] vascular disease. The estimations in this table assume that atherosclerotic accidents (coronary occlusions, apoplexy, etc.) will not happen in the normal course of events.

* Necrotizing arteriolar disease occurs with N retention [illegible] Table 38, p. 300

adequate and when blood viscosity is normal. The relationships of cardiac output, blood viscosity and peripheral resistance in pathological states are discussed in Chapter 2. The fundamental factor which must be constantly considered in any evaluation of circulatory hemodynamics is whether or not the supply of blood to the tissues is adequate to meet their metabolic needs.

This discussion will be primarily concerned with *elevation of blood pressure above 90 mm. Hg diastolic and 140 systolic* when cardiac output is relatively normal, blood viscosity is relatively normal, and circulating blood volume is normal or possibly moderately increased. Other vasopastic states will be considered as they apply to hypertension.*

Patients otherwise in good health who show variable blood pressure elevations which at times are at hypertensive levels *and at others are at* normal ones are considered to be "pre-hypertensive" or to have that constitutional make up which predisposes them to hypertension—unless, of *course, a definite pathological condition can be demonstrated of which the* elevated blood pressure is merely one manifestation. The reader will do well, however, to bear in mind the difference between *stages* in the course *of a condition and the types of diseases which cause that condition.* Confusion in this respect is apparent in many writings on the subject.

HYPERTENSIVE VASCULAR DISEASE

Hypertensive vascular disease is defined as a disease characterized by pathological changes in the arteriolar walls which can be seen microscopically. Usually it develops as a result of chronic hypertension. Obviously severe hypertensive vascular disease can exist for short periods of time with a normal blood pressure (as in hemorrhage or acute coronary occlusion) but this occurs at the expense of blood flow through narrowed arterioles.

STAGES IN THE COURSE OF HYPERTENSIVE VASCULAR DISEASE

Benign hypertension is usually not benign in the long run, often causing serious and fatal secondary pathological changes, but is defined as that stage of hypertensive vascular disease which is only slowly progressive.

Mild benign hypertension is a relatively asymptomatic condition characterized by few detectable pathological changes according to ordinary clinical tests. The blood pressure falls to normal levels with rest.

Moderate benign hypertension is a further stage in development with beginning pathology. The blood pressure falls to normal levels in sleep induced by heavy sedation.

* These limits are arbitrary. A diastolic pressure constantly above 85 mm. Hg may predispose to the later development of hypertension. But just as any single numerical value is difficult to interpret as normal or pathological (just what are the limits of fever, for example?) so an arbitrary value must be assigned below which is normotension, above which is hypertension. The pathological state, however, cannot be inferred from knowledge of a single function of the body.

vasospasm the stages therefore are third approximations of a condition secondary to many primary physiological and pathological aberrations which cause chronic vasospasm and elevation of arterial pressure It must be borne in mind that the factors causing hypertension first operate chronic hypertension then appears which leads after variable period of time to hypertensive vascular disease the latter condition proceed at variable rates of progress usually causing serious pathology occasionally altering into a malignant course rarely ...

appearing so static as to be compatible

7) Single or even multiple determinatio...

made on ambulatory patients cannot be used to determine the stage of the condition No arbitrary limits of systolic or diastolic pressure can be set Observation and evaluation of the whole patient and the whole process must be used in order to estimate the stage and observation over considerable time to estimate the rate of progress

PRIMARY AND SECONDARY HYPERTENSION

The word essential is one which has evolved into a semantic refuge from the realities of ignorance Idiopathic or the tongue-twister agnogenic are more honest expressions of ignorance of etiology and initiate a mental uneasiness which may serve as a challenge to investigation into causes The clinical types of hypertension will be designated by appropriate terms which reflect the present state of understanding whether they are right or wrong can only be decided in the future Fishberg called ...

... therefore discarded the use of the word for many years preferring to designate more on possible etiological and pathogenetic principles than on unconsidered vague terminology

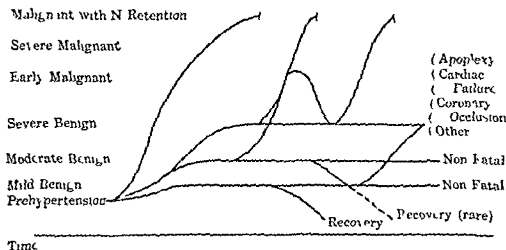
The words primary and secondary hypertension have also been used the first to designate those conditions dependent upon no known cause and the second hypertension dependent upon known organic diseases These terms will be employed sparsely for the exact causes of secondary hypertension are not well understood and may be similar to those of primary hypertension in most cases There is enough suggestive evidence however that the hypertension which ...

... hence it is based ... and organic alterations in several systems of the body all of which can be considered secondary Dexter's modification[3] of our original classification[2] has been remodified in an attempt to clarify the whole subject If confusion has become worse confounded we can only point to the paucity of fundamental knowledge of the whole subject and justify our efforts as the best that can be done up to the present time after many years of deliberation

3

Severe benign hypertension is a stage in which pathological changes in heart, cerebral vessels, or kidneys are readily detectable by ordinary methods (electrocardiograph, x-ray photographs, urinalysis, studies of renal function, physical examination). The blood pressure *does not* fall to normal levels under heavy sedation or anesthesia. All gradations are apparent.

Malignant hypertension is a stage in the course of hypertensive vascular disease, usually associated with renal parenchymal disease, which is characterized by a "fixed" or relatively unvariable high diastolic pressure, signs of diminution of renal function, and especially exudative and hemorrhagic lesions in the ocular fundi with papilledema. The course is rapidly downhill, and renal and/or cardiac failure the terminal event. *Early malignant hypertension* is the *beginning* stage of this alteration, when hemorrhages and/or exudation appear *first in* the fundi. It is sometimes reversible by ordinary non-specific medical measures (rest, sedation) but



[illegible] of the various courses of untreated hypertension and [illegible] [illegible]idents indicated. The total [illegible]

often it progresses to *severe malignant hypertension* and then to renal insufficiency with retention of nitrogen in the blood, uremia and cardiac failure. The term is used to denote a change in the course of hypertensive vascular disease which can cause serious *and* fatal consequences and which demands intensive therapy (Table 3).

These, then, are *the* various stages in the course of hypertensive vascular disease *before* the *condition itself* becomes subjugated to more serious secondary pathological changes in heart, brain or kidneys. They are enumerated *for* prognostic purposes, poor as prognosis may be in any chronic disease where rupture or occlusion *of major* vessels or viscera can occur at any moment. These stages are *in no* way to be considered as disease entities or *types* of hypertension, but merely represent a general evaluation of the degree of progress which hypertensive vascular disease has made. The disease itself is a secondary manifestation of chronic

Chapter

4

HISTORICAL

In the conceit of our assumption of knowledge we are inclined to forget the understanding which pioneers in medical science had of both physical and physiological principles Unhindered by a mass of conflicting experimental facts they were free to propose and humbly accept simple explanations for simple phenomena Observation theory and intuition played a large part in their thinking as the numbers of investigators grew so the various opinions and conclusions increased while the basic observations became covered by incidentalia In some cases however truths suspected by pioneers have begun to emerge slowly and painfully through masses of paper to be rediscovered anew with enthusiasm by modern investigations Retrospect may therefore be rewarding

The oldest reference to arterial disease though coincidental occurs in Exodus VII 3 And I will harden Pharaoh s heart and multiply my signs and my wonders in the land of Egypt The mummy reputedly of that *Pharaoh* Mnephtah whose heart was hardened many times against Moses and the children of Israel showed advanced aortic calcification While we cannot reconstruct the pathology as coronary artery d e a n

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Hypertension i not a new dise e The development of the sphygmomanometer in the middle of the last century led to its wide recognition but the consequences of high blood pressure were known for many centuries Apoplexy is an old disease but its causation by hemorrhage was probably first recognized in modern times by John Jakob Wepfer in 16 He describes the case of a monk aged about forty five years who was afflicted with the gout He was little inclined to an

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atherosclero ... sus of left ventricular hypertrophy recognized for many years were especially described by Laennec in 1819 These are—a strong full pul e strong and obvious *pulsation of the heart* ...

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Extensive clinical use of the sphygmomanometer followed the development of von Basch s (1876) Riva Rocci s (1896) and von Recklinghausen s (1901) instruments and it was only shortly thereafter (1904) that Rom ... erosis thereby

... tewas in 1904
He recognized most of the pathological effects of permanent elevation of arterial pressure many of the possible causes, the associated physiological disturbances and the possibility of multiple factors operating etiologically,

The concept of essential hypertension has been well put by Fishberg ' *The concept of essential hypertension includes those cases of chronic hypertension which neither clinically nor anatomically can be demonstrated to have evolved from antecedent inflammatory disease of the kidneys or urinary obstruction* This definition is very seriously defective in that it defines solely by exclusion but in our present ignorance of the actual etiology it does not seem feasible to define es ential hyperten ion in any more satisfactory way *The very term essential hypertension is a confession of ignorance- and this is its chief virtue* The noun expresses the dominant clinical manifestation and the adjective serves the double function of forewarning of our ignorance of the cause of the hypertension and differentiating it from nephritic hypertension All that the term es ential hypertension really means is non nephritic hypertension The concept of essential hypertension is merely a stop-gap necessitated by our present ignorance

Therefore the condition on which we are reporting may be regarded either as a single disease the cause of which is unknown or a gro p of separate disease with ...
each of
respects ... ost clinical signs ar ... symptoms were once disease *entities* (dy pnea apoplexy gly cosuria hematuria fever edema *obesity* phthisis) which advancing knowledge and clinical observation has catalogued divided and co-ordinated into their proper pathogenetic categories The group represented by the concept essential hypertension is no exception

It is very *important* to discover whether or not non nephritic hypertension was common, or indeed existed at all before the Industrial Revolution Most of the recorded cases were associated with primary renal disease and apoplexy heart failure and uremia ma ...
complication ...

... common in winter and ... the rainy seasons and when the barometer is very low

are constant, and it is not uncommon to find the disease in persons who have none of them The pulse, in particular, is very deceptive, being almost as frequently weak as strong in such cases In this disease the patient experiences more constantly than in any other the sensation of the action of the heart but he is less subject to violent attacks of palpitation irregularity and intermission of the pulse are uncommon There is rather increase of the power of the ventricles than of the noise produced by their action"

Coronary arterial disease, so often consequent to hypertension, was described by Krehl in 1740, and its relation to angina pectoris by Fothergill in 1776 and Jenner shortly before Morgagni (1761) was well aware of the relationship of cerebral vascular disease and apoplexy He was the first to recognize the hereditary nature of apoplexy, describing a patient whose father had died of it and whose uncle had died of a stone in the bladder the patient suffered from both conditions He also remarked upon two brothers both of whom had apoplexy We do not know whether or not he was aware of the autopsy findings on Marcello Malpighi who died in 1694 of apoplexy secondary to renal hypertension (see Chapter 15)

Richard Bright mentioned the thickening of the larger renal arteries in the disease which bears his name and Johnson (1852) first described renal arteriolar sclerosis believing it to be caused by muscular hypertrophy *Gull and Sutton (1872) however were the first to consider the* lesions as the result of hyaline degeneration and fibrosis as good an analysis considering the methods at their disposal as most modern contributions The *pathological background of chronic arterial hypertension* was thus established

Physiological concepts which are yet the subject of argument began in 1830 after the development of the first useful sphygmomanometer by Herisson but not until fifty years later did von Basch investigate hypertension and its relationship to cardiac hypertrophy and hardening of the arteries He was the first to establish the normal standards which are much used today Extensive physiological investigations by Ludwig, Marey and Traube had laid the groundwork which he applied to disease Traube (1856) and Kirkes (1857) were probably the first to show that elevated blood pressure might be the result of high intra-arterial tension although an increase in the action of the heart was then considered the primary cause Traube considered that hypertension was a result of arteriosclerosis but might be the cause of renal hypertrophy *In the minds of most clinicians* however hypertension was associated with chronic Bright's disease until Mahomed first recognized (1874-1881) the condition which was later named *essential hypertension He advanced the ultra modern view that* hypertension *causes* the renal vascular affections found at necropsy

Allbutt (1895) was also ultra modern in his concept He recognized *hyperpiesia* as a condition independent of chronic nephritis and arteriosclerosis and believed that the existence of elevated intra arterial tension might bring about both arteriolar and arterial disease the latter has only recently been proven experimentally Huchard (1893) simultaneously advanced the idea of a generalized angiosclerosis involving heart arteries veins and capillaries

therefore that an abnormally high pressure cannot exist permanently unless there has been some damage to the regulating power of the visceral circulation Permanent high blood pressure cannot be maintained without hypertrophy of the left ventricle

The concept of essential hypertension has been well put by ... cases of chronic hyper... can be demonstrated to ... the kidneys or urinary ... obstruction This definition is very seriously defective in that it defines solely by exclusion but in our present ignorance of the actual etiology it does not seem feasible to define essential hypertension in any more satisfactory way *The very term essential hypertension is a confession of ignorance—and this is its chief virtue* The noun ... manifestation and the adjective ... of our ignorance of the cause ... from nephritic hypertension ... really means is non nephritic hypertension The concept of essential hypertension is merely a stop-gap necessitated by our present ignorance

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Persons who have much fat and a short neck, who disregard the rules of *temperance in eating and drinking make up most of the subjects of bloody* apoplexy They are also exposed by an hereditary disposition and are between the ages of forty and sixty years (Translated from Fourth French edition)

Although some chemical and considerable physiological information has been gathered in the past fifty years, the concepts which were first advanced to explain the physiopathology of hypertension are still being debated in clinical scientific circles, and no new ones of note have been proposed The theory that hypertension might be caused by a change in the quality of the blood which increased the resistance to flow in the small vessels first proposed by Richard Bright in 1836, is the subject of much of our researches

"The obvious structural changes in the heart have consisted chiefly of hypertrophy with or without valvular disease and what is most striking out of 52 cases of hypertrophy (in 100 cases of renal disease), no valvular disease whatsoever could be detected in 34, but in 11 of these 34 more or less disease existed in the coats of the aorta still however leaving 22 without any *probable organic cause for the marked hypertrophy generally* affecting the left ventricle This naturally leads us to look for some less local cause, for the unusual efforts to which the heart has been impelled and the two most ready solutions appear to be, either that the altered quality of the blood affords irregular and unwonted stimulus to the organ immediately or, that it so affects the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system"

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Chapter

5

BIOLOGICAL FACTORS—FUNDAMENTAL TRAITS

As yet neither hereditary traits nor constitution have ever been defined in more than the broadest terms. Chromosomal transmission of distinct traits in animals has been inferred from studies on relatively simple organisms and on plants, but the process is little understood.

HEREDITY

The inference that a predisposition to hypertension, like blond hair, facial characteristics or metabolic defects, can be inherited appears only from statistical analysis of families (Tables 4 and 5). Probably the most convincing work is that of Hines, who attempted to assess the presence or absence of vascular hyperreactivity in families of hypertensive patients. Response of blood pressure to a standard stimulus of pain (the *hand held in* ice water for one minute) was the method used. Vascular hyperreactivity to cold as well as to the stressful influence of the first examination was found to predispose to hypertension (Table 6). His results *are recalled to* mind as a significant contribution to the subject which has been partly neglected in recent years. Objections to the method have *been raised*; obviously not all individuals respond to, or feel pain to the same degree; the test is not significant in many cases of hypertension; not all hyperreactors develop hypertension. If we consider, however, that hypertension may be the result of two infl...

and an environment...

of Hines' work beco...

Those extremists...

the development of the human individual (thereby opposing many centuries of experience by breeders of animals and plants) have argued that a

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... upon the structure of

... onmental influences

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on the other hand are likely to be largely influenced by environmental factors

The weight of evidence at present seems to indicate that in the modern environment of the United States the chances are 4 in 10 for a child with one hypertensive parent to develop hypertension and 9 in 10 for a child both of whose parents are hypertensive. Considering the incidence in the general population, and assuming that environmental stresses will not alter

TABLE 5 —FAMILIES SUBJECT TO HYPERTENSION

Author	No families	Parents hypertensive	No generations	No hypertensive descendants	Remarks
Rosenbloom[11]	1	Both	1	8/10	Parents died of apoplexy at 45
de Vaz for Nikitit[12]			3	3?	2 3 had different terminal events
Hurst	1	Both	3	16??	Nephritis
Budd *	1		3		Bright s disease
Pel[14]	1		4	19	Nephritis
Badia	4		5		Only one case without family history
Allbutt †	1	One	4	4	Paternal ancestors all died of apoplexy
Mortensen *	1	Both	1	10/10	All had apoplexy

* Quoted by Eason & Smith[13]
† Quoted by Fishberg[8]

TABLE 6 —PREDISPOSITION TO LATER DEVELOPMENT OF HYPERTENSION

Author	No cases	Lower limit of hypertension mm Hg	First systolic pressure mm Hg	Per cent later: 10 yrs	Per cent later: 20 yrs	First diastolic pressure mm Hg	Per cent later hypertensive: 10 yrs	Per cent later hypertensive: 20 yrs
Transient hypertension with stress of examination								
Hines[8]	1,5??	160 100	100	0	0	70	0	0
			110–119	? 1	4 9	70 74	3 4	1 4
			120–129	6 1	12 6	75–79	2 4	4 2
			130 139	10 7	33 8	80–84	2 2	? 0
			140–149	35 7	63 1	85 89	? 0	57 3*
			150 160	60 ?	78 4	90–94	41 ?	65 0
						95–100	0 2	56 0

* Prehypertensive range

Author	No cases	Lower limit of hypertension mm Hg	Age at first examination	Rate/1000 developing hypertension	Ratio of hypertension with/without prior transient hypertension	Remarks
Levy et al	?41	150 90	25–29	2 4	4 8	Army officers followed until retirement
			30 34	2 5	2 8	Transient hypertension at first examination
			35–39	5 6	3 7	
			40–44	11 2	4 3	
			45–49	22 0	3 8	
			50–54	33 2	3 4	
			55–59	49 0	3 2	

was aggressive nervous, dynamic and had feelings of inferiority Others have reported both of the twins hypertensive (Table 7) A pair of Siamese twins illustrated several factors One had had renal calculi and the other apparently had not, in the first hypertension was quite severe, in the other labile Their mother had hypertension Their personalities differed the one with the more severe hypertension being thin, nervous and excitable

TABLE 4 —HEREDITY AND HYPERTENSION

Author	No Families / Subjects	Incidence of hypertension in children of hypertensive parents—Per cent: Both parents normotensive	One parent hypertensive	Both parents hypertensive	Incidence of hypertension in parents of hypertensive patients: No patients	Per cent	Remarks
Ayman[1]	277/1524	3 1	28 3	45 5			
Hines[2]	30/250		43 4	95 0			Hyperreactors to cold pressor test or hypertensive
Hines[3]	/54		44 5*				Studied 20 years after first examination
	/44	7					
Barach[3]	58			69 6	31	50	Usual family history
						95	Careful family history
O Hare et al[4]					300	68†	Also in 37 6% of 437 controls and 37 5% of 128 over 40 years
Schroeder and Steele[15]					46	41 0	Neurogenic hypertension only
					50	64 0	Organic renal disease only
Bechgaard[7]					634	28 7	71 3% various other causes
Weitz[3]					82	76 8‡	Also in 30 3% of 267 control Predisposition inherited as a Mendelian dominant
Rautmann[3]					53	57 7	59% of children with slightly higher pressure had positive family histories 10% of controls also

* Positive family history
† Vascular diseases all types in parents
‡ Parents and sibs

the other being calm placid and stout Inadequate studies have been made of children raised by foster parents whose heritage was known The extensive study now under way by Thomas [8] who is following the careers of medical students may throw some light on the question of hereditary predisposition to disease Certainly it is as easy to believe that disturbances such as vagotonia or sympathotonia are inherited as that they spring full grown like Pallas Athena from the nervous system of the individual without ancestry other than thought and conflict Personalities

hypertensive women may be fat thin slender stocky, blond red haired and brunette we have no figures on the subject except the recollection of those of our patients and not even an impression has arisen in a review of several thousand with the exception of the definitive type discussed later (Chapter 16)

TABLE 8 INTERCORRELATIONS BETWEEN HYPERTENSION AND OTHER DISEASES (DAVIES)

Correlation with following diseases	Hospital service	Number of patients	Both diseases Expected	Both diseases Observed	Correlation	Probability value p*
Coronary Thrombosis	All†	40 490	40	103	Pos	0 001
	Medical	11 0[illegible]	[illegible] 9	117	Pos	<0 000
Asthma	All	40 490	40 2	31	Neg	0 07
	Medical	11 0[illegible]	[illegible]	14	Neg	0 0004
Laennec Cirrhosis	All	40 490	18 8	1[illegible]	0	0 25
Duodenal Ulcer	All	40 4[illegible]0	49 4	34	Neg	0 01
Diabetes Mellitus	Medical	11 0[illegible]	1[illegible] 2	191	Pos	<0 000
Obesity	Medical	11 0[illegible]	101 8	163	Pos	<0 000

I [illegible] that the diseases were several times higher in medical service than in the entire hospital group However trends toward positive and negative correlations with coronary thrombosis and asthma respectively were in good agreement in two series Data taken from admission to Barnes Hospital St Louis Mo 1940 through 19[illegible]

* Values p are calculated by Chi Square analysis and represent the fraction of times a difference as great as that shown between observed and expected value may occur by chance A p value of 0 01 or less is highly significant

† Pediatric service and other patients under twenty years of age not included

A few hints that constitutional predispositions to certain diseases exist are found in the recent work of Davies[37] who studied the intercorrelations of hypertension to coronary thrombosis duodenal ulcer bronchial asthma Laennec's cirrhosis diabetes and obesity The well recognized coexistence of hypertension and coronary thrombosis was proven statistically suggesting either that one influenced the development of the other or that both occurred in the same type of person Curiously enough neither obesity nor diabetes were accompanied by a higher incidence of coronary occlusion than that expected by chance when blood pressure was normal Hypertension and duodenal ulcer tended to be mutually exclusive while hypertension and Laennec's cirrhosis did not Coronary thrombosis occurred in expected rates in patients with duodenal [illegible]

[illegible] two constitutional types are present in hospital population one predisposed to hypertension and the other to ulcer None of the other diseases analyzed showed a clear-cut distribution (Table 8) It is our clinical impression that severe allergic states (bronchial asthma recurrent urticaria eczema) are unusual occurrences in hypertensive patients

PREDISPOSITION TO HYPERTENSION

An analysis of the records of 22 741 U S Army officers was completed during and after World War II by Levy Stroud White and Hillman [illegible]

materially (a false assumption in this changing world) it becomes obvious that hypertension will increase with each generation to the point of affecting *most of the population* Natural readjustments however, will undoubtedly arise to compensate for this inimicable state of affairs in spite of the unfortunate fact (unfortunate from the viewpoint of national heritage) that most hypertensive persons die after the child-bearing period

Table 7—Hypertension in Twins

Author	No twins hypertensive	Remarks
Pechgaard[7]	Both	Perhaps identical
Frohlich[22]	Both	Age 12 identical
Friedman *et al*[23]	One	Personalities very different No family history
Weitz[24]	Both	Identical age 63
Jones *et al*[25]	Both	Siamese The more hypertensive had organic renal disease

Therefore, we may accept the hypothesis that the predisposition to hypertension is probably often hereditary or at least familial and that it is associated with vascular hyper-reactivity until direct evidence offers disproof Whether the ability to react to painful (and emotional) stimuli by vasoconstriction is dependent upon altered function of vasoconstrictor nerves or upon altered chemical or physical structure of the smooth muscle of blood vessels is not known The most careful study that of Weitz[8] suggests that the defect is inherited as a Mendelian dominant *

CONSTITUTION

The "constitution" of hypertensive individuals is even less well understood Constitution is used in the sense of the physical emotional and mental make up of human beings and is therefore psychosomatic in the largest sense of the word Physically hypertensive men are often believed to be of the stocky squarely built sthenic type (with many exceptions) who run to fat emotionally they are dynamic aggressive doers with urges and ambitions often larger than their capabilities Hypertensive women with the exception of one type discussed at length later do not fit into this pattern of physical structure nor are they usually among the most aggressive and masculine varieties of female In our own experience the discovery of a typical hypertensive habitus in the male is an occasion for a mental review of the many who do not fit the pattern a good majority probably are sthenic, and a much greater number of stocky individuals have hypertension than have duodenal ulcers No incontrovertible figures are known which may throw light on this subject Also in our experience

* It is not certain however that all patients must have a family history positive for hypertension Of 24 carefully examined in this regard in which no known hypertension or its sequelae were present in the family 21 died with a malignant course one was under the age of twenty 7 under the age of thirty 16 under the age of forty and all under the age of fifty It is necessary to study family trees of patients of this sort more carefully

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12 LAMON J and SMITH C L M Hereditary and familial nephritis The Lancet 207 633 1924
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17 ...

... Transient hyper various systolic and diastolic

20 ...
21 ...
22 ...
23 ...
24 ...
25 ...
26 ...

27 DAVIES D F Intercorrelations between hypertension and other disease states (to be published)

Predisposition to the later development of sustained hypertension was associated with the finding of transient hypertension at an earlier examination in ratios varying from 2 8 to 4 8 times that *expected, depending upon* age, with the finding of transient tachycardia in a ratio of 3 4, and with the finding of overweight of 20 pounds or more in a ratio of 2 5 If all three factors were present, the chances for a given individual to develop sustained hypertension were 12 5 times as great as if they were absent, and 4 1 times as great for him to retire from service on account of having cardiovascular renal disease Overweight in these statistics did not increase the death rate significantly Hines' figures were therefore confirmed by a study involving another group of subjects (Table 6)

Therefore, it appears that this curious complex of heredity, constitution and environment which is so poorly understood but which predisposes an individual to hypertension may make itself manifest by one or more of the following signs transient hypertension resulting from the stress of an examination (a most variable stimulus) or from the painful stimulus of ice water (a less variable one), transient tachycardia resulting from the stress *of an examination overweight and a certain type of personality Definition* of each of these influences, of their relative weights, and of the necessity of each or all to be present for hypertension to develop awaits further and more careful study

TENTATIVE CONCLUSIONS

1 The predisposition to hypertension is an hereditary trait transmitted as a dominant characteristic

2 If it is not then the predisposition is 'catching from parent to child during the latter's emotional development but not all children catch it In this case, the cardiovascular abnormality must be primarily neurogenic

3 The predisposition is manifested by a reaction to stress by means of vasospasm tachycardia or other neurogenic cardiovascular alterations

4—Constitution in the sense of body build nervous and emotional make-up may be in some cases *especially in men another manifestation of the* hereditary predisposition

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Chapter

6

ETIOLOGICAL FACTORS—ENVIRONMENTAL INFLUENCES

An environment considered adverse by the individual is probably a requisite for the development of "primary" hypertension and may influence adversely the course of "secondary" hypertension. This belief is inferred from what is known of incidence rates in environments other than our own and from careful studies of the personality traits of hypertensive patients (see Chapter 7). Careful studies, however, have not been made to determine the effect of environment upon the development of the condition. Therefore, the best that can be done is to present what figures are available in the United States and what impressions are available in foreign, especially oriental, countries.

INCIDENCE OF HYPERTENSION IN THE UNITED STATES

There is little doubt that the existence of hypertension leads to several serious pathological changes which cause death and disability. There is also no doubt that these hypertension-induced diseases constitute one of the greatest causes of death in the United States. Estimated death rates shown in Table 9 for the United States indicate that over a fifth of all deaths are due directly or indirectly to the results of hypertension. In this it stands foremost, with the exception of arteriosclerosis, as the major killer of our civilization. The apparent paradox that hypertension rarely is fatal in itself is resolved by a consideration of the seriousness of its effects upon heart, brain and kidneys.

Because of the chronic nature of most cases, the incidence in the United States is much greater than the 320,000 yearly deaths would indicate. Opinions differ as to the actual figures. Out of over 40,000 admissions to a group of teaching hospitals in Missouri, both on private and ward services, 15.9 per cent of patients exhibited hypertension; this in spite of the general tendency of physicians to treat hypertension in office practice and admitting officers in teaching hospitals to find other disposition of patients with cerebral accident, uremia and chronic cardiac failure (Table 10).

Probably the most accurate figures were obtained from insurance policy holders, a selected group from a higher economic level than much of the population (Table 11). *These statistics are alarming and probably do not represent true incidences.* However, it is reasonably safe to state that hypertension is the most prevalent serious chronic disease in persons over forty years of age, *probably occurs in half of the population over sixty*, and increases rapidly with age. The distribution between urban and rural

Table [illegible] Hypertension as a Cause of Death (U. S.)

	Cardiovascular Deaths				Deaths from All Causes			
	19[illegible]		19[illegible]		19[illegible]		1951	
	Total (thousands)	Per cent	Total (thousands)	Per cent	Total (thousands)	Per cent	Total (thousands)	Per cent
Hypertension without mention of heart	13	1.7	14	1.8		0.[illegible]		0.9
Hypertension with Heart Disease	84	11.3	83	10.9		[illegible]		[illegible].7
Hypertension and Arteriosclerosis								
Vascular lesions of Central Nervous System	1[illegible]	20.[illegible]	1[illegible]8	20.[illegible]		10.[illegible]		10.6
Myocardial degeneration	70	[illegible]	[illegible]	[illegible].0		[illegible].8		4.7
Total	333	45.3	324	42.[illegible]		23.0		21.8
Arteriosclerotic Heart Disease	323	43.4	338	44.1		22.2		22.[illegible]
General Arteriosclerosis	31	4.2	3[illegible]	[illegible].8		2.2		2.[illegible]
Total	3[illegible]	47.6	375	48.[illegible]		24.4		2[illegible].4
Deaths from All Causes					1,4[illegible]0	100	1,481	100
Total Cardiovascular Deaths					7[illegible]	[illegible]1.4	76[illegible]	51.6
Total Hypertension and Arteriosclerosis		90.8		[illegible].4	687	47.4	[illegible]	[illegible].1

Modified from National Heart Institute Circular No. [illegible] October 19[illegible] reporting statistics from National Office of Vital Statistics

TABLE 10.—INCIDENCE OF HYPERTENSION IN HOSPITAL AND OUT-PATIENT ADULT POPULATIONS

Author	No. cases	Lower limit of hypertension B.P. mm. Hg	Age Distribution*	Per cent hypertension	Year reported	Remarks
Autopsy Cases						
Clawson[1]	30,265	Cardiac Hypertrophy	All ages	8.6	1941	55.5% of non-congenital cardiac deaths
Baggenstoss & Barker[2]	100	150/90 and Cardiac Hypertrophy	All ages	29.0	1941	Non-congenital cardiac deaths only
Shure[3]	1,000	150/95 or Cardiac Hypertrophy	All ages	31.9	1942	
Hospital Cases						
Reisinger[4]	1,369	150/90	All ages	6.0	1937	Male veterans
Tonkin	47,084	150/90	All ages	0.7	1939	General hospital (Pa.)
Davies[5]	10,164	140/90	All ages	15.9	1951	General hospital (Mo.)
Friedman et al[6]	1,006	150/90	30-79	21.7	1942	Surgical patients (Mass.)
Waldt[7]	248	150	Over 60	40.0	1942	Germany
Stone & Vanzant[9]	915	Several	All ages	47.7	1927	Cardiacs only (South)
Master et al[10]	500	Several	All ages	76.5	1939	Coronary occlusions only (N.Y.)
Out-Patients						
Gager[1]	2,000	140-150/90	All ages	11.3	1928	N.Y. public and private
Bowers[2]	461	150/90	33-82	12.0	1929	Private (North)
Schwab & Schulze[11]	10,188	150/95	All ages	9.3	1931	O.P.D. (Tex.)
Wetherby[14]	5,540	150	All ages	19.4	1932	O.P.D. (Minn.)
Vaughan & Graham[1]	4,000		All ages	5.5	1930	Office (South)
Braasch et al[16]	945	145	All ages	20.0	1940	Private (Minn.)
Bechgaard[2]	21,552	160/100	All ages	3.9	1946	Ambulatory (Denmark)

* Adolescents and adults except when indicated.

Note.—Much of the material in Tables 10–14 was abstracted from a summary prepared by John A. Morell, Ph.D., of the Bureau of Applied Social Research, Columbia University, and published.[18]

TABLE II. INCIDENCE OF HYPERTENSION BY AGE (SELECTED GROUPS)

Author	No. cases	Lower limit of hypertension B.P. mm. Hg	Decade of life								Population Standard
			[illegible]	3	4	5	6	7	8	9	
Levy et al.	22,741	150/90		0.6	1.0	1.6	18.5				Army Officers
					2.4	10.7	20.7				(U. S.)
Life Extension[6]	8,3[illegible]	150/100			2.0		18.0	33.0			Periodic Exams (Male)
	5,02[illegible]	150/100			3.0		34.0	58.0			Periodic Exams (Female)
Rusch et al.[6]	5,331	150/[illegible]				5.8	12.[illegible]	26.[illegible]	40.[illegible]	11.6	Merchant Seamen
Willius[7]	248	150/[illegible]						40.0			Patients over 60
Friedman et al.	1,006	150/90			10.2	26.0	34.0	47.5	51.0		
Branch et al.[8]	[illegible]	145	0	2.1	8.4	16.4	28.7	47.4	53.1		Patients Clinic
Master et al.[9]	14,84[illegible]	150/90				[illegible]	40.6	[illegible]	[illegible]		Males (N.Y.)
						32.0	[illegible]	[illegible]	[illegible]		Females (N.Y.)
Monroe	4,085	/90						34.4	2[illegible]	22.7	Male Hospital
								[illegible]	24.2	13.0	Patients
	3,013	90						31.2	34.7	30.[illegible]	Female Hospital
								37.0	32.[illegible]	36.[illegible]	Patients (Boston)

By 5 year period.

TABLE 12.—INCIDENCE OF HYPERTENSION IN YOUNG ADULTS

Author	No. examined	Lower limit of hypertension	Age distribution	Per cent hypertensive Male	Per cent hypertensive Female	Year studied or reported	Population sampled
Boynton & Todd[22]	75 218	140/90	Mean 21.5 M 21.1 F	7.3 6.07	1.4 2.54	1930–42	College Students (Minn.)
Diel & Hessdorffer[23]	155	150	Av. 19.8	20.0		1933	College Students
MacCraken[24]	10 000	140	20–30		5.0	1939	Applicants for Teachers (Mich.)
Palmer[25]	3 598	140	18–25	10.1		1914–18	College Students (Mass.)
Thacher[26]	15 500	150	18–25	3.3		1938–39	College Students (Ill.)
Alvarez et al[27]	14 934	140/	81%<25	20.0	2.7	1918–21	College Students Cal.)
Selective Service	Millions	Hypertension	18–35	1.2		1940–41	Selective Service (Total U.S.)

TABLE 13.—INCIDENCE OF HYPERTENSION IN MALES AND FEMALES

Author	Number of cases Male	Number of cases Female	Lower limit of hypertension B.P. mm. Hg	Age distribution years	Per cent hypertension Male	Per cent hypertension Female	Year examined or reported	Type of population
Boynton & Todd[22]	43 800	31 418	140/90	18–25	7.3 6.07	1.4 2.54	1930–42	College Students (Minn.)
Alvarez et al[27]	6 000	8 934	140	85%<25	20.0	2.7	1918–21	College Students (Cal.)
Symonds[28]	162 356		140/	>16	[illegible]	3.5	1907–1[illegible]	Policy holders
Bowers[29]	169	292	150/90	40–70	10.0	13.0	1929	Office patients
Master et al[30]	8 483	6 366	150/90	40–90	40.9	50.7	1943	Industrial workers and patients
Gager[31]	1 000	1 000	140/90	All ages	12.6	16.1	1928	Clinic patients
Wetherby[32]	2 292	1 259	150/100	All ages	15.1	21.6	1932	Hospital patients
Schwab & Shulze[33]	10 188		150/95	All ages	8.6 3.4	11.1 10.6	1931	Heart disease only

TABLE 11. INCIDENCE OF HYPERTENSION IN NEGRO AND WHITE POPULATIONS

Author	Number of cases: Negro	Number of cases: White	Lower limit of hypertension, BP, mm Hg	Age distribution, years	Percent hypertensive: Negro	Percent hypertensive: White	Type of population
Schwab & Shulz	10 188		1[illegible]0	All ages	6.[illegible]	3.4	Male hospital patients
					14.7	10.6	Female hospital patients
Keesling[a]	2 2[illegible]0	1 [illegible]00	160	All ages	13.[illegible]	4.4	Male prisoners
Allen[a]	1 000		140/90?	Under 40	25.0		Factory workers, male
Orenstein	3 000		140/[illegible]	18–[illegible]	[illegible].8		Selective Service examinees, male
Hornett & Ratcliffe[a]	1 781	[illegible]	140/[illegible]	All ages	18.6	1[illegible].0	Southern farm workers
Weiss & Prudden				Under 40	10.1	3.6	Hospital patients (Louisville)

TABLE 12 – INCIDENCE OF HYPERTENSION IN YOUNG ADULTS

Author	No examined	Lower limit of hypertension	Age distribution	Per cent hypertensive Male	Per cent hypertensive Female	Year studied or reported	Population sampled
Boynton & Todd[22]	75 218	140/90	Mean 21 5 M / 21 1 F	7 3 / 6 07	1 4 / 2 51	1930–42	College Students (Minn)
Diel & Hesdorffer[23]	155	130/	Av 19 8	20 0		1933	College Students
MacCraken[24]	10 000	140	20–30		5 0	1939	Applicants for Teachers (Mich)
Palmer	3 598	140	18–25	10 1		1914 15	College Students (Miss)
Thacker	15 000	150	18 25	3 3		1938–39	College Students (Ill)
Alvarez et al[17]	14 934	140/	84%<25	20 0	2 7	1918–21	College Students (Cal)
Selective Service	Millions	Hypertension	18 35	1 2		1940 44	Selective Service (Total U S)

TABLE 13 —INCIDENCE OF HYPERTENSION IN MALES AND FEMALES

Author	Number of cases Male	Number of cases Female	Lower limit of hypertension B P mm Hg	Age distribution years	Per cent hypertension Male	Per cent hypertension Female	Year examined or reported	Type of population
Boynton & Todd[22]	43 800	31 418	140/90	16 25	7 3 / 6 07	1 4 / 2 51	1930–42	College Students (Minn)
Alvarez et al[17]	6 000	8 934	140	84%<25	20 0	2 7	1918–21	College Students (Cal)
Symonds	162 376		140	>16	5 7	3 5	1907 19	Policy holders
Bowers	165	292	150/90	40 70	10 0	15 0	1929	Office patients
Master et al	8 453	6 366	150/90	30 90	10 9	10 7	1943	Industrial workers and patients
Unger	1 000	1 000	140/90	All ages	12 6	16 1	1928	Clinic patients
Wetherby	2 282	3 258	150/100	All ages	15 1	21 6	1932	Hospital patients
Schwab & Shulz	10 188		150/95	All ages	8 6 / 3 4	11 1 / 10 6	1931	Heart disease only

TABLE I. INCIDENCE OF HYPERTENSION IN [illegible] COUNTRIES [illegible] ORIENT

Author	No. of cases	No. died hypertension	No. with elevated B.P.	Lower limits of hypertension B.P. mm. Hg	Per cent with elevated B.P.	Per cent with hypertension	Race
[illegible]	1 000	1	20	160	0 5	0 0	Chinese patients [illegible] Hosp. China, Changsha (Yale)
	2 8			140/	0 0		Healthy Chinese, Changsha
	1 0			140/	0 0		Healthy Whites, Changsha
	102†			140/	3 0		White U. S. soldiers, Philippines
	366†			110/	0 0		Philippine soldiers, Philippines
	[illegible]			110/	[illegible] 0		Foreigners, Philippines
	#			140		0 0	Canton [illegible] Philippines
Tung	1 233	3		160			Chinese males (over 40 years [illegible])
Harris, Houston					Rare		Chinese [illegible]
Donnison	1 800	0	[illegible]	150	Very rare	Unknown	China; African Negro [illegible] Kenya Colony (2 cases of elevated B.P. [illegible])
[illegible]	103	0	0	150	0		Australian aborigines [illegible] age 50+
[illegible]	142	0	0	140/90	1 2		[illegible] Kimos ([illegible]) age 40 [illegible]
[illegible]	500	1		160/100		0 2	Patients in Oriental [illegible] Hospital. Rare in India, Iran, Indonesia, Malaya (all types of [illegible])
[illegible]	3 000	—			10 0		[illegible] private practice (age 30–50)

Including [illegible] and [illegible] Five [illegible] in [illegible] hospital patients from [illegible] accidents including [illegible]

† Quoting Chamberlain

‡ Quoting Musgrave & Sison

Quoting Cadbury

Quoted by [illegible]

populations has not been well studied but incidence rates may be higher in the former. It has been found impossible to compare various occupations (i.e., government Civil Service employees with steady jobs, progressive advancement and assured retirement, and ambitious business executives whose security is that of opportunity) because of the importance of the age factor which is not taken into account in various studies. However, it may be less common in Army Officers than in insurance policy holders in New York.

Hypertension is, however, not uncommon in young adults in the United States if we accept the rigid criterion of 140 mm. Hg systolic and 90 mm. Hg diastolic pressures as the upper limits of normal (Table 12). Such figures are always subject to the criticism that the methods of examination, the amount of emotional stress imposed upon the subject, his position and many other variables may have varied from one study to another. It is interesting in this respect that under the demands for manpower during World War II the incidence of "high blood pressure" in young males was low, reflecting perhaps the attitudes of the examining physicians of draft boards.

Comparative statistics are always difficult to evaluate because of the strong age factor, but if we can trust them two more trends appear. First, hypertension is more common in young males than in young females in the total population and especially that over forty the reverse is true, hypertension being more common in women. All studies but one bear this out (Table 13). Second, hypertension is more common in American Negroes than in American whites. Although the number of studies is few, the results appear fairly conclusive (Table 14). This difference may be environmental, for hypertension is said to be rare in the African Negro.[1] *

Because of late recognition of hypertension and its direct effects, no one knows whether the incidence rate is increasing rapidly or has been fairly static during the past fifty years. The growth of an older population *promotes a relative increase in incidence, but the absolute rate by decades* has not been compared with previous generations. All impressions, however, favor the belief that more people are suffering from hypertension and a greater percentage of the population over age forty exhibits it than thirty or sixty years ago. The statistics are in the main inaccurate and definite conclusions lacking, but one would have expected much more attention in the older literature if hypertension had been as prevalent in the nineteenth century as it is in the middle of the twentieth.

INCIDENCE OF HYPERTENSION IN ORIENTAL COUNTRIES

The studies available are few and far between which can settle conclusively the question, is hypertension a disease of Western Civilization. Caution must be exercised in the evaluation of any data bearing on a dis-

* The only reference found suggesting a high incidence of hypertension in native African negroes was unavailable and therefore unchecked. Hartnett and Ratcliffe[39] quote Dubois as giving an incidence of 35.5 per cent in 200 Congo natives, the degree of civilization being unstated. Their own figures comparing two rural groups, white and negro, in Mississippi, cannot be evaluated for age incidence.

TABLE I. INCIDENCE OF HYPERTENSION AND RELATED CONDITIONS ACCORDING TO STATEMENTS OF LOCAL PHYSICIANS IN VARIOUS TROPICAL FOREIGN CITIES (1953)

City	Country	Hypertension: Essential	Hypertension: Both renal disease	Arteriosclerosis	Apoplexy	Clinical Standards	Climate	Remarks
Baghdad	Iraq	Uncommon		Fairly rare		Fair	Hot dry	British run hospital
Kermanshah	Iran	Rare	Uncommon	Rare		Fair	High Temperate	American Mission Hospital
Teheran	Iran	Unknown	Uncommon	Rare	Uncommon	Fair	High Temperate	American Mission Hospital
Srinagar	Kashmir	Rare	Uncommon	Uncommon		Good	High Temperate	British Mission Hospital
Lahore	Pakistan	Unknown	Uncommon	Uncommon	Uncommon	Excellent	Tropical	British Medical School
Calcutta	India	Rare	Uncommon	Uncommon		Excellent	Tropical	British Hospital
Colombo	Ceylon	Rare†	Uncommon	Uncommon	Uncommon	Fair	Wet Tropical	Native Medical School
Singapore	F M S	Rare	Uncommon	Fairly frequent‡		Excellent	Wet Tropical	British Hospital
Batavia	N E I	Rare	Uncommon	Uncommon		Excellent	Wet Tropical	Dutch Medical School

* Coronary artery disease almost unknown
† One case encountered personally in 124 hospital patients examined
‡ Coronary artery disease common in Chinese
Note All Hospitals and Medical Schools had European trained men on medical and pathological services

case with such a strong age factor as hypertension when one compares countries with vastly *different life expectancies* (U. S. about sixty in 1930, India about twenty-one). However, all of the evidence points to the rarity of hypertension among Oriental peoples, Australian aborigines,* Eskimos and Negroes in Africa (Table 15). We can accept much of it as true without needing to know the exact figures.** A personal experience

TABLE 16.—INCIDENCE OF CARDIOVASCULAR RENAL DISEASES IN 500 HOSPITAL MEDICAL CASES

(Personally examined[10] in five general hospitals in tropics)*

Disease	No. cases	*Per cent of cases*	*Remarks*
Essential Hypertension	1	0.2	Young male Singalese
Chronic Myocarditis	12	2.4	Mostly undiagnosed cardiac insufficiency
Generalized Arteriosclerosis	1	0.2	Only true case seen
Hemiplegia	3	0.6	
Acute Nephritis	5	1.0	Under age 20, only 1 in native
Nephrotic Stage	3	0.6	Over age 20, all in natives
Chronic Nephritis & Uremia	12	2.4	*All in native*
Nephrosis, Lipoid	7	1.4	Fulfilled classical definition of Volhard (in China)
Pyelonephritis	4	0.8	Including perinephritis
Toxemia of Pregnancy	0	0	
Acute Rheumatic Fever	5	1.0	
Rheumatic Heart Disease	10	2.0	
Total Renal	31	6.2	
Total Cardiac	27	5.4	
Total Possibly Hypertensive	9	1.8	Only 1 with severe hypertension

* Lahore, Calcutta, Colombo, Singapore, Batavia.

in the Orient in 1933 proved to the author the rarity of hypertension, coronary disease, arteriosclerosis and apoplexy while attesting to the frequency of acute and chronic glomerulonephritis and rheumatic heart disease. The actual statistics, which *depend upon European trained* clinicians and pathologists for their validity, are presented for what they may be worth. At least they show that rheumatic heart disease is more common than hypertensive (*Tables 16–19*), *a marked inversion of the situation* in the United States.

These different rates of morbidity in countries having vastly different civilizations from that of our own require explanation. We do not believe that the much lower life expectancies prevalent in the so-called "backward" countries account for the differences in comparative morbidities but rather that hypertension is a disease of civilization as we know it with its attendant strains and stresses, competitive urges, complexities of living, and *repetitive impacts*. These *shifting stresses have removed many*

* A careful study was made by Nye[28] of 65 male and 40 female aged aborigines from Cape York Peninsula, Australia. No hypertension was found. The subjects were meat eaters, heavy users of strong tobacco, were under emotional tension when the measurements were made, and lived in a communal society.

** Raab[1] has made a survey of the distribution of hypertension up to 1932, based on published reports and impressions. *In Europe the incidence was said to be approximately* the same or a little less than in the United States. It was said to be low or absent in the Arctic, Africa, China, East Indies, India.

strongly against the emotional tensions which can induce hypertension than a host of other factors Only in the Western European races has man developed techniques for explaining and controlling the forces of an adverse nature in so doing he has built himself an environment so complex ... ted for living that he survives only at the ... tension

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Atherosclerosis may develop more rapidly when cert ... are common than when they are not when localized atherosclerosis may precipitate hypertension in individuals so predisposed (See Chapter 15 p 287) Obesity itself may predispose to hypertension a careful study by Bechgaard[1] indicates that one-half of hypertensive patients suffered from considerable excess weight and only 3 per cent were below normal One-third however were of normal weight Obesity in hypertensives how ... of a common perso al ... l fact an en locrine

Diets vary the world over high and low fat animal and vegetable fat high and low protein—the one constant feature is a high carbohydrate content which may come from rice wheat or potatoes Within our own civilization diets vary according to individual custom The human body has marvelous adaptability the diet of a ...

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of us from the daily contacts with nature and the spiritual aspects conducive to a peaceful compromise with the business of living found in less rapidly advancing countries. The philosophical acceptance of the in-

TABLE 18. INCIDENCE OF KIDNEY DISEASES IN AUTOPSY RECORDS PERSONALLY EXAMINED (1933)

Anatomical diagnosis	Colombo (1000 cases)†		Batavia (5000 cases)‡		Lahore (500 cases)#	
	No. cases	Per cent	No. cases	Per cent	No. cases	Per cent
Chronic Glomerulonephritis	6	0.6	3	0.1	79	15.8
Contracted Kidney	17	1.7	34	1.1	0	0
Large White Kidney	6	0.6				
Small Granular Kidney *	10	1.0				
Parenchymatous Nephritis		0.5				
Interstitial Nephritis	3	0.3				
Tubular Nephritis	1	0.1				
Uremia	4	0.4				
Nephrosis			[illegible]	0.2		
Total	52	5.2	43	1.4	79	15.8

* Possibly hypertension
† Quality of record fairly good (British supervised)
‡ Quality of record excellent (Dutch)
Quality of record excellent (British trained pathologist)

TABLE 19. INCIDENCE OF RHEUMATIC HEART DISEASE, ACUTE RHEUMATIC FEVER, ACUTE AND CHRONIC NEPHRITIS IN SIX LOCALITIES IN TROPICS (1933)[49]
(Per thousand medical admissions corrected)

Disease	Teheran	Lahore	Calcutta	Colombo	Singapore	Batavia
Scarlet Fever		None	None	None	None	None
Acute Rheumatic Fever	[illegible] 5	[illegible]	3.2	[illegible]	0.5	0.9
Rheumatic Heart Disease, Chronic						
Clinical	7.8			7.4	0.8	[illegible]
Pathological		[illegible]		[illegible]		9
Acute Nephritis	0.67		[illegible] 4	3.7	8.3	1.0
Chronic Nephritis						
Clinical	7.3		[illegible] 4	13.7	[illegible] 1	4.6
Pathological		158		5[illegible] 0		14.3

evitable by the Chinese, the dependence upon firmly established rules and routines by the Indian,* the deep-rooted unhesitating mores of the African Negro, the unconcern with the future of the Malay (there is no future tense in the language), and the humble resignation of most primitive and prolific races towards events beyond their control may militate more

* According to Dr. H. Chakravarti of Calcutta (personal communication 1952), the incidence of hypertension is extremely low in the villages of Bengal. However, when one compares persons of the same age and economic status, the clinical impression is that the incidence in these groups is almost as high in Calcutta as in the United States. Physicians and lawyers, for example, are frequently victims of coronary occlusion. Autopsy figures are unreliable for this aspect, since most postmortem examinations are made on unclaimed bodies from the lowest economic groups. Therefore, the more civilized the individual, the greater are his chances for developing hypertension, regardless of climate, diet or race.

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of tampering with natural foods or starvation is present, can we ascribe ills to foods or their lack. There is a vast unexplored territory here as well as many known facts to be applied, but within the grounds of present knowledge there is no evidence that human hypertension is caused by high protein diets, high fat diets, not enough vitamins or carbohydrates from the wrong source (nor prevented by eating rice, fruit, nuts or grasses). A definite correlation with intake of specific foods remains to be proven.* The use of diets to influence diseases already firmly established is, of course, another matter.

While the protein-eating Negro, Eskimo, Sikh and Moslem and the vegetarian Chinese and Hindu may both show little hypertension, other factors must be considered to account for its rarity in some races and frequency in others. (1) Climate is one unexplored factor—although the climates of the plateau of Iran, the mountains of Kashmir, and the northern coast of China are similar to our own. (2) Racial hereditary or endocrine differences, which have not been measured, suspected or demonstrated, but which may exist in the fundamental make-up of the individual. (3) The simplicity of life and the absence of nervous strain. Foster[34] who lived in China, and Nye[35] in Australia, incline to the last factor as the most important. Extensive epidemiological investigations are needed to settle the question, which is etiologically of the highest importance.

TENTATIVE CONCLUSIONS

We may draw the following tentative conclusions from the figures as presented.

1. Arterial hypertension is a condition the incidence of which increases rapidly with advancing age, becoming significantly prevalent in the fifth decade. It accounts directly or indirectly for over a fifth of deaths from all causes in the United States.

2. Without a breakdown of incidence according to age, no two groups of the population can be compared as to various environmental influences unless the disease is virtually absent in one group.

3. In the United States, hypertension occurs more frequently in young men than in young women; in later life the relative incidences are reversed.

4. In the United States, Negroes are more prone to develop hypertension than whites.

5. In Oriental countries, hypertension is uncommon or rare. In the African Negro, Australian Aborigine and native Eskimo it is probably rare.

6. Hypertension appears to be a disease of Western Civilization in so far as statistics and impressions are accurate.

* One exception in experimental animals may be significant. Olsen has found that rats made deficient of vitamin B6 by de-oxypyridoxine develop hypertension quite rapidly.[36] Also we cannot exclude the possibility that preserved foods used by highly civilized races contain trace metals which alter enzymatic systems in kidney or blood vessel.

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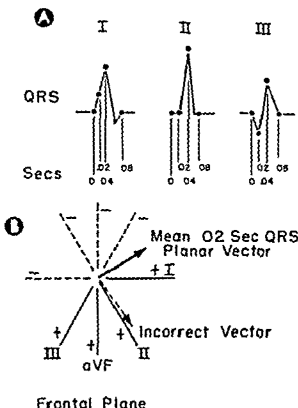


Fig 31 —Construction of mean 0 02 second vectors from the routine electrocardiogram In A the QRS complexes are registered in leads I II and III with corresponding points (measured from the actual onset of ventricular activation) on the deflections identically numbered and separated from each other by 0 02 second intervals It is evident that at 0 02 second after *onset of the QRS interval lead II* registers zero potential so that the mean vector for the initial 0 02 second period is perpendicular to the lead axis of II as shown in B If the above leads were recorded consecutively rather than simultaneously the phasic relationship indicated in the figure would not be apparent *Hasty inspection of the three leads might lead to the conclusion* that point 2 in lead II occurs at 0 second and point 3 at 0 02 second after onset of depolarization Thus the mean 0 02 second QRS vector might be thought erroneously to parallel the positive lead axis of II As far as the extremity leads are concerned this error can be avoided by application of Einthoven s law For example the error of identifying point 3 in lead II as the 0 02 second deflection is evident because the corresponding deflections in leads I and III when added (Einthoven s law $I + III = II$) yield a zero value instead of a positive value for the voltage of the 0 02 second deflection in lead II Unfortunately no such mathematical relationship exists between the deflections recorded by the precordial leads and so there is no way to compensate for the lack of phase relationship in these leads

tor with its perpendicular transitional plane The vector can be rotated freely within the cylinder except at its fixed point which in the model is equivalent to the electrical center of the heart Urschel and Abbey have attempted to quantitate the information obtainable from vector models by mounting degree scales in the bottom of the cylinder (for the horizontal plane) and on the side of the cylinder (for the sagittal plane) The hexaxial reference figure is depicted on a plastic surface behind the cylinder

The QRS complex can also be divided into 0 02 second portions which can then be treated in each of the standard limb and precordial leads as individual deflections From these values can be derived a mean spatial vector for each 0 02 or 0 04 second interval of the QRS complex The construction of mean vectors for such short intervals of the QRS deflection is likely to involve significant errors unless leads are recorded simultaneously at increased paper speed (Fig 31) This problem will be considered in more detail in a later section concerning the construction of vector loops from the electrocardiogram (see pp 102–104)

Theoretical Bases of the Electrocardiographic and Vectorcardiographic Leads

EQUIVALENT DIPOLE AND SEMIDIRECT LEAD HYPOTHESES

THE SINGLE EQUIVALENT dipole theory which has been developed in the preceding chapters proposes that in terms of body surface potentials (*a*) the electrical field produced by heart muscle can be represented at any instant by a single equivalent dipole and this in turn by a mean instantaneous spatial vector and (*b*) the voltage registered in any given lead is directly proportional to the projection of the instantaneous vector on the axis of the lead but is inversely proportional to the cube of the distance between the dipole and lead electrode. This concept implies that the scalar deflection registered by an electrocardiographic lead—whether it is an extremity, precordial, back *or* esophageal lead—merely reflects the manner *in which the lead taps* a single dipole vector. The validity of the single equivalent dipole concept is fundamental to vectorcardiography and to the vector method of electrocardiographic interpretation. Although vectorcardiography in its present form appeared soon after the development of the cathode ray oscilloscope in the 1930s the vector concept and its implications were known to Einthoven and associates and to some of his contemporaries. In fact Einthoven utilized lead axes and the vector projection method to determine the mean electrical axis (vector) of the scalar deflections recorded in leads I, II and III. The extremity leads were considered compatible with Einthoven's hypothesis and the equivalent dipole theory because the points of application of the lead electrodes to the body surfaces were accepted as being "electrically remote" from the heart. However with the development of the precordial leads a new concept emerged which has been variously termed the *semidirect lead concept* or the *localized regional* or *proximity potentials concept*. This theory stressed the relative proximity of the precordial lead electrodes to the heart and was based on Wilson's observations in animal experiments that unipolar precordial leads and leads which were applied directly to the epicardial surface of the heart recorded QRS complexes quite similar except for size. From this experimental observation was derived the concept that the potential variations of a precordial lead reflect to a great extent the potential variations of the epicardial surface closest to the lead electrode. Thus because of their apparent similarity to direct leads the unipolar precordial leads were designated *semidirect leads*.

The semidirect lead or proximity potentials hypothesis was next extended to include the unipolar limb leads whose potential variations were thought to be influenced chiefly by the potential variations of the surface of the heart they faced. The corollary to this proposal was that the potential variations of the unipolar limb leads must necessarily be governed by the position of the heart in the chest. Conversely if the configuration characteristic of the lead deflection recorded from each different aspect of the heart is established then the electrocardiogram should indicate the "electrical" position of the heart. This concept carried with it the implication that anatomic heart position and rotation could be determined electrocardiographically but as will be indicated later in both theory and practice the semidirect lead hypothesis is probably of limited validity in light of more recent studies. The results of some of these studies are in brief as follows

1 Schmitt, Levine and Simonson have carried out electrocardiographic mirror pattern cancellation

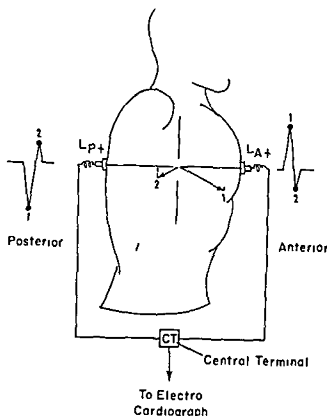


Fig 32 —Simplified illustration of the manner in which electrocardiographic mirror pattern cancellation studies are performed If the dipole concept is *correct and the electrical forces of the heart can be* represented by instantaneous resultant vectors then for a lead such as lead L_A whose exploring electrode is placed on the anterior surface of the chest there must be another lead L_P on the opposing surface of the back which records QRS complexes having a mirror image relationship to those recorded by lead L_A Thus vector *1* projects positivity on lead L_A and equal negativity on L_P Vector *2* projects negativity on lead L_A and equal positivity on lead L_P These two leads are unipolar leads since their indifferent or negative electrodes are attached to a central terminal (*CT*) For this reason their lead axes pass through the dipole center Inasmuch as the two leads register mirror image deflections their positive lead axes lie on the anterior and posterior portions of a single line extending through the dipole center Their mirror image potentials cancel out each other when passed into the central terminal so that theoretically zero voltage would be recorded from the latter If localized potentials significantly influence the electrocardiogram anterior chest lead L_A would be dominated by potentials from the anterior surface of the heart and lead L_P would respond mainly to potentials from the posterior aspect of the heart In consequence mirror patterns could not be recorded and cancellation of potentials would be impossible

studies designed to prove or disprove the dipole theory (Fig 32) *In normal subjects and patients with* heart disease they measured potential variations from electrode sites connected to form an electrical bridge circuit This circuit was so arranged that potentials referred from two electrode sites to a central terminal could be canceled out if the heart behaved as a true dipole Along a line passing through the dipole position the magnitudes of electrical potential referred to a central terminal have a fixed distribution increasing as the distance from the dipole lessens changing polarity or sign as the dipole is passed and then decreasing as the distance from the dipole becomes greater Therefore leads recorded from the opposite points of *this line when it is extended to the body surface* should exhibit mirror patterns of each other if the dipole theory is valid In normal subjects and patients *with myocardial infarction* Schmitt Levine and Simonson found that on the average only 9% of any pair of mirror patterns failed to cancel There were poorer degrees of cancellation in patients with bundle branch block and this was attributed to migration of the cardiac dipole In brief the results of the studies by these investigators were in agreement with the dipole theory The influence of localized potentials on the electrocardiogram was thought to be a very minor *one Frank and other investigators have performed* similar studies with results almost identical to those above

2 If the dipole theory is correct it should be possible to derive from the vectorcardiogram scalar lead deflections which resemble those actually recorded at the various precordial lead points The electrical and manual methods for deriving scalar leads are discussed later The lead electrodes of the vectorcardiograph are relatively remote from the heart compared with the electrodes of the unipolar precordial leads Therefore if local potentials play a significant role in the genesis of the precordial electrocardiogram the *deflections in the scalar precordial leads as derived* from the horizontal plane vectorcardiogram should differ from those actually recorded while the deflections should be similar if the dipole concept is valid Milnor and his co workers and Grishman and Scherlis Duchosal and Grosgurin and others have found the resemblance between derived and recorded scalar lead deflections to be quite close even in the case of the diagnostic Q waves of myocardial infarction According to the semidirect lead hypothesis the abnormal *Q waves produced by myocardial infarction*

result from the transmission of negativity from the left ventricular cavity through a "window" of necrotic myocardium and are therefore a manifestation of the semidirect nature of the precordial leads However scalar lead deflections derived manually or electronically from vectorcardiograms recorded (with electrically remote leads) in instances of myocardial infarction show diagnostic Q waves This common observation would seem to indicate that the Q waves of infarction are representative of a general disturbance in the balance of electrical forces which in turn leads to the appearance of mean instantaneous QRS spatial vectors of abnormal direction and/or magnitude

Although these two conflicting theories continue to be a source of controversy the weight of evidence at present is preponderantly in support of the validity of the equivalent dipole theory when applied to body surface potentials and surface leads Some authorities admit the validity of this evidence only insofar as it pertains to the extremity leads but they retain the semidirect lead hypothesis as the explanation of the manner in which the unipolar precordial leads respond to the cardiac forces Occasional reference will be made in this text to the semidirect lead approach to the electrocardiogram particularly in the section on electrical heart position and cardiac rotation (pp 60–64) but otherwise the text will adhere strictly to the equivalent dipole or resultant vector concept

LEAD VECTOR VECTOR IMAGE AND LEAD FIELD CONCEPTS

Let it be supposed that a hypothetical experiment is being performed to determine the relationship between the voltage recorded in a lead and the orientation of the dipole axis with respect to the anatomic axis of the lead (Fig 33) A plaster model of the body torso has been filled with saline solution and electrodes have been attached at positions on the model corresponding to the left shoulder (L) right shoulder (R) and the junction of the left leg with the trunk (F) R ...

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therefo ... e anatomic axes of these leads can be considered to form an equilateral triangle An artificial dipole vector mounted on a pivot has been placed in the mod l

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actual cardiac ... in the human chest The experiment will consist of (a) rotating the dipole

... quilateral triangle hypothesis is entirely valid the following results would be expected (a) At the time each lead registers its maximal deflection the dipole vector should be parallel to the anatomic axis of the lead—along the 0 −180 axis for lead I the +60 −120 axis for lead II and the +120 −60 axis for lead III (b) The magnitude of the maximal deflection in each lead should be equal since the artificial dipole is of constant strength and its projection on each lead axis is maximal and therefore the same

However it is found that the maximal deflection is recorded in lead I when the artificial dipole vector points superio ...

... again instead of lead I recording a deflection of resultant zero voltage as would be anticipated the lead registers a negative deflection It becomes evident from these findings that the lead voltage can be correlated with the orientation of the dipole vector only if one assumes that in addition to its anatomic axis lead I has an effective axis directed differently The effective axis of lead I can be visualized as an imaginary line drawn parallel to the dipole vecto ...

... anatomic axes Thus even though the experiment has not yet been completed it can already be seen that the effective lead axes of the bipolar limb leads form a scalene or Burger tri... rather th ...

reasons

of the ... model although other factors such as the location of the lead electrode the irregular contour of the torso model and the finite size of the model as a volume conductor (and in the human torso additional variables such as the uneven conductivity of the body tissues) would influence the orientation of the effective lead axes

The second unexpected finding in the hypothetical

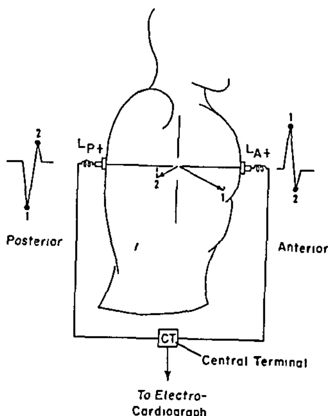


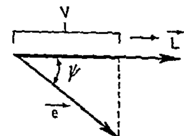
Fig 32 —Simplified illustration of the manner in which electrocardiographic mirror pattern cancellation studies are performed If the dipole concept is correct and the electrical forces of the heart can be represented by instantaneous resultant vectors then for a lead such as lead L_A whose exploring electrode is placed on the anterior surface of the chest there must be another lead L_P on the opposing surface of the back which records QRS complexes having a mirror image relationship to those recorded by lead L_A Thus vector *1* projects positivity on lead L_A and equal negativity on L_P Vector *2* projects negativity on lead L_A and equal positivity on lead L_P These two leads are unipolar leads since their indifferent or negative electrodes are attached to a central terminal (*CT*) For this reason their lead axes pass through the dipole center Inasmuch as the two leads register mirror image deflections their positive lead axes lie on the anterior and posterior portions of a single line extending through the dipole center Their mirror image potentials cancel out each other when passed into the central terminal so that theoretically zero voltage would be recorded from the latter If localized potentials significantly influence potentials from the posterior aspect of the heart In consequence mirror patterns could not be recorded and cancellation of potentials would be impossible

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experiment is that the magnitudes of the maximal deflections in leads I II and III are different Evidently the leads must be weighted differently in terms of their lengths The concept of lead *weight* or length was shown previously to apply to the bipolar and unipolar limb leads By calculations based on the Einthoven equilateral triangle it was possible to demonstrate that the bipolar lead axes are longer than the axes of the augmented unipolar limb leads while the

since the dipole vector parallels the lead vector when the maximal deflection is recorded Therefore the above equation can be restated as follows *The size of the maximal deflection in a given lead is proportional to the magnitude or length of the lead vector or the voltage equivalent or value of each unit length of the projected component of the dipole vector is proportional to the length of the lead axis* Thus the lengths of the lead vectors of leads I II and III in the experi



$V = \vec{L} \cdot \vec{e}$ or

$V = \bar{L}\,\bar{e}\,\cos\psi$

$\vec{L}$ — LEAD VECTOR

$\bar{L}$ — MAGNITUDE OR LENGTH OF LEAD VECTOR

$\vec{e}$ — DIPOLE VECTOR

$\bar{e}$ — LENGTH OF DIPOLE VECTOR OR DIPOLE MOMENT

ψ — ANGLE SUBTENDED BY LEAD AND DIPOLE VECTORS

V — PROJECTION OF DIPOLE VECTOR ON LEAD VECTOR OR VOLTAGE PROJECTED ON LEAD

Fig 34 —Relationship of the l ad d flect... ing and accompanying equa... sion of the projection of the c...

axes of the Wilson unipolar leads are the shortest of all It was also shown that, with reference to the magnitude of a cardiac vector the unit of amplitude in the augmented unipolar leads is equivalent to only 87% of an equal deflection in the standard bipolar leads and the percentage drops to 58% in the case of the Wilson unipolar leads

Like the extrem... polar leads also have t... direction (r... be represer... vector quantities or lead vectors The directions of the lead vectors have already been established in this experiment To *determine the* relative lengths of these lead vectors a concept by Burger and van Milan can be utilized (Fig 34) This proposes that the deflection in a given lead is the dot product of the dipole vector and lead vector and can be calculated by multiplying the magnitudes of these vectors by the cosine of the angle (ψ) they subtend, or $V = \vec{e} \cdot \vec{L} = \bar{e}\,\bar{L}\cos\psi$ In this experiment the m... nitude of the d... be ignored,

ment can be made proportional to the magnitude of the maximal deflection in each of these leads

With the information provided by the experimental observations it is now possible to construct a triangle *R L F'* having as its sides the lead vectors of leads I II ...

... image *point* of *L* Apex *R* lies inferiorly and anteriorly to *R* the site of the right shoulder electrode and is the image point of *R* while *F* is the image point of *F* In fact for every point on the surface of the torso model there is a corresponding image point in other words the anatomic torso surface can be represented electrically by an image surface According to Frank who advanced this theory of the *image vector* the projection of the cardiac dipole vector on a second vector originating at the dipole center and extending to a point on the image surface multiplied by the length of this vector gives a result which is proportional to the true unipolar potential measured at the corresponding point on the torso surface If an electrode is relatively near the di

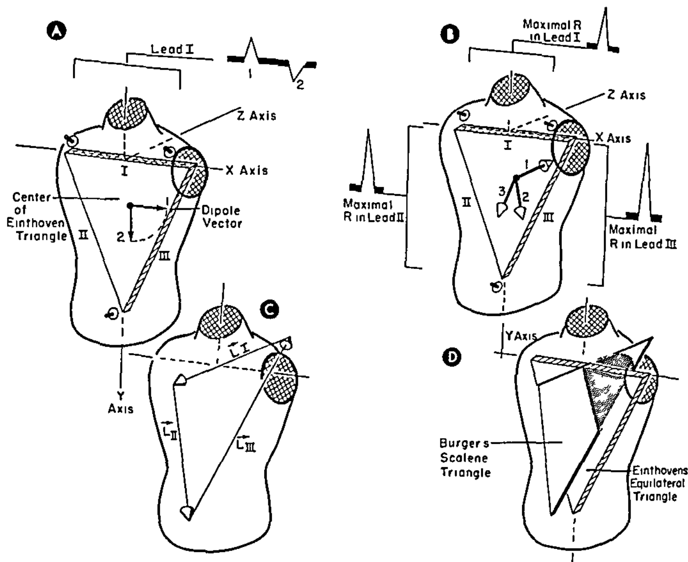


Fig 33 —Schematic diagram of an experiment with torso model and artificial dipole illustrating the basis of the lead vector concept In **A** with the dipole vector at position *1* lead I records an R wave of unexpectedly low amplitude in

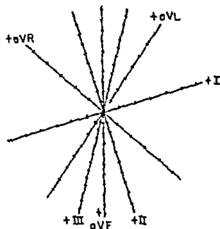
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dipole vector *1* and the effective posterior and superior to right anterior and inferior (**C**) In a similar manner the directions of the lead vectors of leads II and III can be determined from the directions of the dipole vector when it projects maximally on lead II (vector *2*) and on lead III (vector *3*) In **B** it should be noted that the maximal R waves for leads I II and III are not equal in size Consequently the three

of leads I II and III is a scalene triangle which does not lie entirely in the frontal plane



Fig 36 —Four nonequilat ral triangles repre senting variations in the Burger triangle as the result of differing locations of the dipole center in a torso model (After Langner)



Millimeters per Scale Unit			Proportionality Factors
Leads	I	7 5	1
	II	4 5	0 6
	III	3 75	0 5
	aVR	8 25	1 1
	aVL	6	0 8
	aVF	4 5	0 6

Fig 37 —Hexaxial reference figure formed by lead vectors calculated from torso model studies The effective directions of the various lead axes are shown and the differing lengths or magnitudes of the lead vectors have been corrected for by the use of proportionality factors Thus, since th lead vector of lead III is twice the length of that of lead I it is assigned a proportionality factor only one half that of lead I As a result, the scale unit of lead III is one half the length of that of lead I

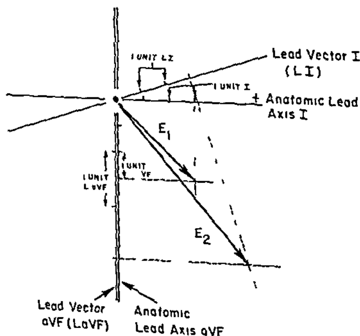


Fig 38 —Reference figure consisting of the anatomic lead axes of I (labeled *lead I*) and aVF (labeled *lead aVF* the line on the right in the vertical double line) and the lead vectors of lead I (*lead vector I* or L I) and of aVF (*lead vector aVF* or L aVF the left line of the vertical double line) The anatomic lead axes are marked off in equal scale units and for convenience the scale units in lead vector I are the same as those in the anatomic lead axes Since lead vector aVF has a length or magnitude approximately twice that of lead vector I its scale units are twice as large as those in the other axis If the lead vectors had the same direction and magnitude as their corresponding anatomic lead axes a cardiac vector 1 (E) projecting equally on both would be accurately reproduced in the vectorcardiogram or could be accurately constructed from the electrocardiographic deflections in the two leads However because of the differing magnitudes of the lead vectors and the altered direction of lead vector I there is an exaggeration of the vertical projection of the cardiac vector in the frontal plane vectorcardiogram and the electrocardiographic deflections recorded by these leads as indicated by vector 2 (E)

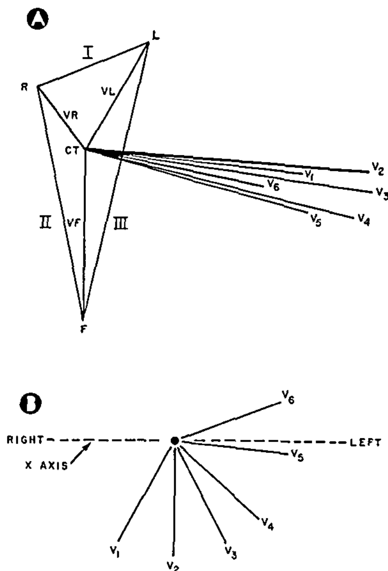


Fig 35 –Lead vectors of the conventional twelve electrocardiographic leads **A** the lead vectors of the standard bipolar and unipolar limb leads (frontal view) as *calculated from data obtained from torso* model studies The lead vectors of the unipolar precordial leads originate from point *CT (Wilson s central terminal) The* precordial lead vectors are visualized in the frontal plane in order to demonstrate their respective magnitudes and their angles of declination from the horizontal **B** the positions of the precordial lead vectors in the horizontal plane shown in relation to the transverse axis (X) representing the intersection of the frontal and horizontal planes of the body The magnitude of these lead vectors is not indicated in **B** (After Helm)

pole its image point is far from the dipole if the electrode is situated far from the dipole the image point is near the dipole

The closely related concepts of the lead vector and image vector are actually based on studies which resemble although in a more complicated form the hypothetical experiment described in the preceding paragraphs *At present these concepts are being used* to study the electrical qualities of the body as a volume conductor and the effects of dipole eccentricity One of the most promising applications of the lead vector and image vector concepts has been suggested by the investigations of Burger and van Milaan Frank Helm and others As already indicated in the hypothetical experiment described above it has been shown that the Einthoven triangle is not equilateral but is scalene or nonequilateral (Burger triangle) because of the differing lengths and directions of the bipolar lead vectors Attempts have been made to calculate Burger triangles for the human body and to calculate the lead vectors of the other routine leads (Fig 35) While data obtained so far is not definitive it seems likely that reasonably accurate Burger triangles will eventually be constructed possibly for each type of body habitus (Fig 36) At present however it is felt that the use of the average scalene *Burger triangle such is that constructed from the* data of Frank and Kay probably introduces less error in most subjects than the conventional Einthoven triangle It is worthy of note that Einthoven s law (Lead I + lead III = Lead II) is applicable to the Burger triangle A revised hexaxial reference figure for the frontal plane based on the lead vectors has been calculated from torso models (Fig 37)

In order to calculate more *accurately the various* frontal plane vectors from the electrocardiogram the differing lengths of the lead vectors can be corrected for by means of proportionality factors These are ob

system (X Y and Z axes of the body) This type of lead system called an orthogonal lead system was thought to offer some advantage over systems based on the Einthoven triangle However if these rectangular systems are reconstructed using the appropriate lead vectors their geometric configurations can also be shown to be distorted to varying degree

Another approach to the problem of devising more satisfactory leads has been that of McFee and Johnston These investigators utilized models of selected planes of the body through which water was made to flow in sheets The models were so constructed that the course of flow encountered resistances analogous to the electrical resistances in the body Thus by causing the water to flow between points corresponding to different electrode positions the flow lines could be mapped out for combinations of points These flow lines were considered to approximate the lines of current flow in the body so that the pattern of flow lines for a given lead represented the lead field for the lead (Fig 41) McFee and Johnston are now attempting to devise improved leads by selecting combinations of electrodes in which the undesirable features of the flow lines tend to cancel out one another

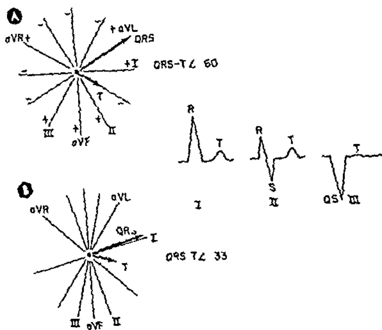


Fig 40 —The influence of the different directions of the lead vectors as opposed to the anatomic lead axes the magnitude variations in the leads being ignored From the schematic complexes labeled leads I II and III it can be seen that the mean QRS vector is perpendicular to the lead axis of II and the mean T vector is perpendicular to the axis of lead III If these vectors are constructed in the ordinary hexaxial reference figure (A) the QRS-T angle obtained is 60 which is borderline abnormal However if these vectors are plotted in the reference frame (B) formed by the effective axes of the various leads the QRS-T angle resulting is only 33 a value well within normal limits

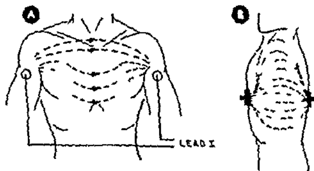


Fig 41 —Pattern of current flow lines or the lead field of lead I (A) and of an anteroposterior bipolar chest lead (

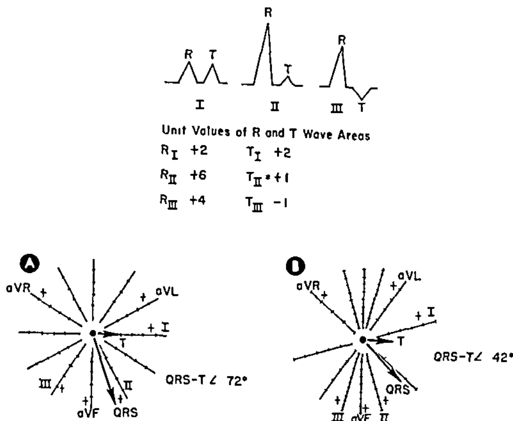


Fig 39 —Application of the corrected hexaxial reference figure to the calculation of the frontal plane QRS–T angle It will be assumed in this figure that a planar QRS–T angle greater than 60 means that the corresponding spatial QRS–T angle also exceeds the upper limit of normal of 60 Similarly a planar QRS–T angle less than 60 implies that *the spatial QRS–T angle is also less than 60 In actual fact this assumption is usually a valid one in the interpretation* of normal and abnormal electrocardiograms

In A the mean QRS and T vectors are plotted on the usual hexaxial reference frame based on the equilateral Einthoven triangle (The vectors are constructed from the relative values for the areas of the diagrammatic QRS and T complexes in leads I II and III given above) *The frontal plane QRS–T angle obtained is approximately 72* and therefore is abnormally wide

If however the r [illegible] correct for the differing lengths [illegible] ing frontal plane QRS–T

1 amplitude unit = 4 mm

1 scale unit in **A** and in lead I of **B** = 5 mm

The scalar units of the remaining leads in **B** are obtained by multiplying the proportionality factors of the leads times 5 mm

tained by dividing the length of lead I by the length of every other frontal plane lead The proportionality factors are used to mark off properly the scale on each lead axis so that the longer the lead vector the smaller are its scalar units

The following correction values have been calculated by Langner

Lead I 1	Lead aVR 1 1
Lead II 0 6	Lead aVL 0 8
Lead III 0 5	Lead aVF 0 6

It is evident from the above proportionality factors that the lead vectors of II III and aVF are about twice as long as the lead vector of lead I Thus in the electrocardiogram there tends to be an exaggeration of the vertical component of the heart vector (Figs 38 39 and 40)

Like the preceding scalar leads the vectocardiographic leads also are modified by the shape dimensions and conductivity of the trunk and by the electrode positions Thus the anatomic lead axes and corresponding lead vectors of the vectorcardiographic leads differ in length and direction For example since the Einthoven triangle is not equilateral systems of electrode placement based on it must necessarily have lead vectors differing significantly from the anatomic lead axes Several of the lead systems have been so constructed as to have their anatomic lead axes parallel to the axes of the natural co ordinate

THE P WAVE

Unlike the ventricles the atria do not have a specialized conduction apparatus Instead the excitation wave initiated by a sinus impulse fans out radially from the sinoatrial node and spreads uniformly in this manner through the atrial muscle The elementary electrical forces produced by activation of the atria vertically downward so that the P wave in lead I becomes smaller and the P waves in leads II III and aVF larger On the other hand in short stocky individuals the frontal orientation of the mean P vector may approach the 0 axis or positive axis of lead I in which case the P wave in lead I tends to be larger than

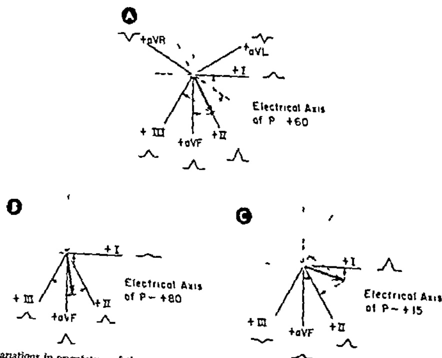


Fig 43 —Variations in orientation of the mean manifest electrical axis of P (Â P) In A ... vector or mean manifest electrical axis of P (Â P) ...

ca ... vectors radiating in all directions from the sinus node However the net effective or average direction of these instantaneous P vectors in the frontal plane—in other words the mean manifest electrical axis of P (Â P) or the frontal mean P vector—approximates the +60 axis of the frontal hexaxial reference figure or the positive half of the axis of lead II (Fig 43) Consequently the P waves tend to be tallest normally in lead II isoelectric or diphasic in lead aVL and upright in leads I and aVF Inverted P waves are normally present in lead aVR In normal subjects of tall and slender body habitus the frontal mean P vector is sometimes oriented more usual and the P wave in lead III may be diphasic or inverted

The horizontal plane mean P vector is relatively variable in orientation ranging from 0 to +60 ... in gener ... slightly ... figure ... inverted ... of lead ... Inasmu ... are almost inv ... atria th ... show ...

With the completion of atrial depolarization the

The Normal Electrocardiogram

GENERAL SEQUENCE OF CARDIAC EXCITATION

THE SPREAD OF EXCITATION through the heart follows a sequence which is largely determined by the site of origin of the excitation impulse, the anatomic distribution of the specialized conducting pathways, and the speed of conduction in the different parts of the heart. The sinoatrial node, which is located in the wall of the right atrium near the entrance of the superior vena cava, normally is the most rhythmical focus in the heart and therefore is the site of origin of the excitation impulse and the dominant cardiac pacemaker. Sinus node discharge per se produces no detectable deflection in the electrocardiogram, although the sinus impulse initiates atrial activation, the latter

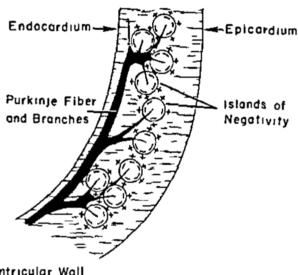
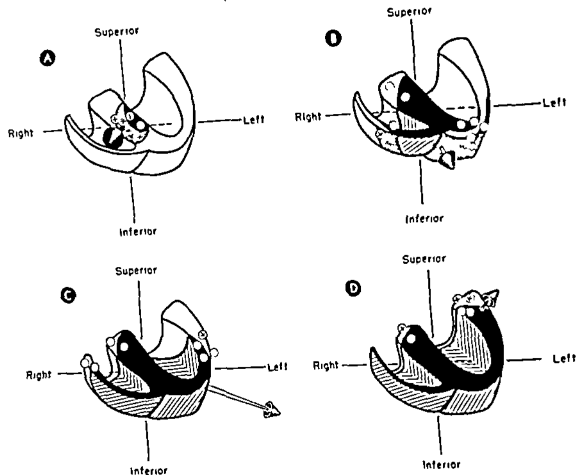


Fig 42.—Schematic section of the ventricular wall showing the Purkinje fibers shortly after onset of ventricular depolarization. Since the Purkinje fibers penetrate the inner two thirds of the ventricular wall, excitation begins as scattered *islands of negativity* at the termini of the Purkinje fibers. Measurable activation potentials are not

producing in turn the electrocardiographic P wave. In the course of its spread through the atrial myocardium, the excitation impulse arrives at the atrioventricular node. This structure consists of a bundle of *longitudinal conducting fibers which merge inferiorly* with the common bundle of His situated in the upper portion of the interventricular septum. The atrioventricular node picks up the atrial excitation impulse and transmits it to the intraventricular conducting pathways. The bundle of His is relatively short and soon divides into two main branches—the left and right *bundle branches—which descend subendocardially on* either side of the interventricular septum. The bundle branches terminate by branching profusely to form an extensive network of conducting fibers, the Purkinje system, which is distributed widely throughout the subendocardium of both ventricles and the lower two thirds of the interventricular septum.

The subendocardial spread of excitation through the myocardial syncytium takes place about 5–10 times more rapidly than its subsequent radial passage outward through the ventricular wall. In addition, substantial evidence accumulated by various investigators supports the belief that the subendocardium and inner layers of ventricular myocardium are normally electrocardiographically silent during depolarization. As an explanation of this finding, it has been suggested tentatively that the Purkinje fibers may extend more deeply into the inner ventricular wall than previously supposed (Fig 42). This may result in the inner ventricular wall being activated too rapidly and in too many directions for any measurable potentials to be produced. Thus, in effect at least, the electrical forces arising during ventricular depolarization are generated almost entirely by the outer subepicardial one third of the ventricular wall.

ventricular w



nterior (C)
activation
leted depo-

branches The excitation process then passes rapidly over the endocardial surfaces of both ventricles and shortly thereafter begins to spread transmurally through the apical walls of the left and right ventricles

✓ 0 02 second apicoanterior VA vector —Within 0 02 second of onset of the QRS interval the activation wave has passed through the lower two thirds of the septum but it continues to spread in an apex to-base direction over the right septal surface and basal one third of the septal myocardium Meanwhile the activation wave front has penetrated to the epicardial surface of the apical right ventricular wall and is now moving through the lateral wall of the right ventricle and through the apicoanterior wall of the left ventricle The rapid extension of the excitation process through the apical myocardium is probably due to the relatively thin layer of muscle in this region or perhaps to the deeper penetration of the Purkinje fibers into the apical myocardium The electrical forces produced by activation of the free walls of the left and right ventricles can be represented by two vectors directed to the left and right of the long axis of the heart Since the left ventricular forces and their representative vector are of greater magnitude than their right ventricular counterparts the resultant of the two vectors which is called the 0 02 second apicoanterior VA vector in this text is oriented along the long axis of the heart—i e anteriorly to the left and somewhat inferiorly (Fig 44 B) The frontal plane projection of the apicoanterior vector is directed approximately along the +60 axis or the positive half of the axis of lead II If the 0 02 second VA vector is superimposed on the frontal and horizontal reference frames it projects positivity on leads I V_6 aVF and V_1 and V_2 causing all leads but V_2 to inscribe the upstroke of an

downstroke of the P wave reaches the isoelectric line. In cases of complete heart block or atrioventricular dissociation it is sometimes possible to discern a negative deflection following the P wave. This deflection represents the atrial T (repolarization) wave and is called the Ta (or Tp) wave. When seen it is usually directed oppositely to the P wave. In most instances, however, the Ta wave is buried in the following QRS complex. With rapid heart rates the Ta wave can cause an apparent depression of the ST segment.

The P–R Interval

The P–R interval *extends from the beginning of the P wave to the onset of the first component of the QRS complex* (Q or initial R) and usually does not exceed 0.20 seconds in normal adults. After inscription of the P wave the base line of the electrocardiogram usually remains isoelectric inasmuch as the electrical potentials formed during the remainder of the P–R interval are of relatively insignificant magnitude. The P–R interval is used primarily *as a measure of the conduction time* through the atrioventricular node, although in fact it represents the total time required for the impulse to travel from the region of the atria near the sinus node to the ventricles. This apparent discrepancy between fact and clinical usage arises because the atrioventricular node has such a very slow rate of conductivity that it constitutes the main conduction bottleneck between sinus node and ventricles. Thus the major portion of the P–R interval is required normally *for passage of the impulse through the atrioventricular node.* While changes in intra-atrial conduction time can also alter the P–R interval, such changes are infrequently encountered in contrast with disturbances of atrioventricular conduction.

THE QRS COMPLEX

In the following paragraphs the spread of depolarization through septal and ventricular myocardium and the electrical forces of differing direction and magnitude thereby produced will be schematized in terms of a series of hypothetical instantaneous cardiac spatial vectors. These vectors will here be designated *ventricular activation vectors* (VA vectors) to distinguish them from manifest vectors determined from the electrocardiogram or vectorcardiogram and from vectors representing actual cardiac forces (as opposed *to those recorded*). *However, the direction and relative magnitude of each of these VA vectors conform* more or less to the characteristics of comparable instantaneous vectors in the normal vectorcardiogram and to the facts known regarding the course of ventricular activation. These VA vectors are assumed to be resultant or mean vectors, each representing the average direction and magnitude of all electrical forces produced at a given instant by the heart, and for this very reason they can be related only in a general way to activation of a specific region of the heart.

Since the original investigations of Lewis and Rothschild many years ago, septal activation has been thought to occur first on the left septal surface and thereafter to spread as a double wave of envelopment from above downward and from without inward. This concept of septal depolarization has been modified [illegible]

0.01 second septal VA vector.—An isopotential line is written *in the electrocardiogram while the excitation* impulse is passing through the atrioventricular node and the common bundle of His. The left septal surface is the first to be activated and this occurs in its upper third because of the early ramification of the left bundle branch in this region. Inasmuch as the septum lies relatively parallel, not perpendicular, to the frontal plane, the VA vector representing this phase of septal depolarization is directed anteriorly as well as to the right (Fig. 44 A). In intermediate *and vertical spatial positions of the heart* the septal vector is thought to pass upward; in horizontal heart position, to pass downward. Purely septal depolarization is exceedingly transient, probably not much longer than 0.01 second in duration. If this septal vector were to be projected on the extremity and [illegible]

V_6, which normally record [illegible] and (*b*) *initial positivity* in lead V_1, which usually displays a small initial R wave. Either a small Q wave or positive deflection may be written in lead aVF, depending on whether the septal vector is directed somewhat superiorly or inferiorly.

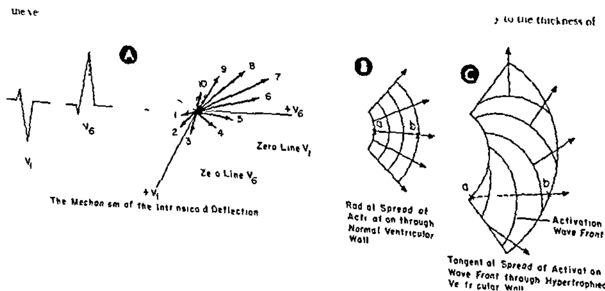
After onset of septal depolarization, the excitation wave spreads rapidly *over the entire left septal surface* and at the same time it emerges on the right septal surface initially near the base of the anterior papillary muscle where the right bundle gives off its first

second VA vectors would project maximal positive voltage on leads V_1 and V and the 0 04 second VA vector would do the same in the case of lead V_6 Thus for each lead from V_1 to V_6 the instantaneous vector projectin, maximal positivity tends to appear later and to project greater positive voltage Not only is this circumstance the basis for the normal transition from resultant QRS negativity in right precordial leads to resultant positivity in left precordial leads but it is also responsible for the different times of onset of the intrinsicoid deflection in leads V_1 and V_6 as will be explained later (Fig 46)

In the normal precordial electrocardiogram the change from resultant QRS negativity to resultant positivity can occur gradually so that one or more intervening leads record *transitional* or *equiphasic RS deflections* The precordial lead registering the transitional RS deflection is termed the *transitional lead* Not infrequently the transition from resultant negativity to positivity takes place abruptly between two adjacent leads in this case the transitional lead can be visualized as situated *intermediate between* the two leads *in* question

One of the concepts which evolved from the semi direct lead hypothesis was that rotation of the heart on its longitudinal axis could be recognized by certain changes in the precordial QRS transition It was thought that the transitional RS deflection was recorded by the precordial lead whose exploring electrode was situated over the interventricular septum Lead electrodes to the right of this point were believed to record primarily right ventricular potentials and electrodes to the left left ventricular potentials If the transitional QRS complex was registered by a

Fig 46 –The present concept of the intrinsicoid deflection The time of onset of the intrinsicoid deflection in leads V and V is measured from the onset of the QRS deflection in each lead to the points where the perpendicular (dashed) lines dropped from the peaks of the R w ... as to the nature ... are representativ... during the QRS ... the ve... to the thickness of



R wave (Fig 45) In the case of lead V_1, the 0 02 second VA vector contributes to the downstroke of the R wave since the positive voltage projected on V_1 by the 0 02 second vector is usually of lesser magnitude than that produced by the initial septal vector

0 04 second left ventricular VA vector —At about 0 04 second after onset of septal depolarization the

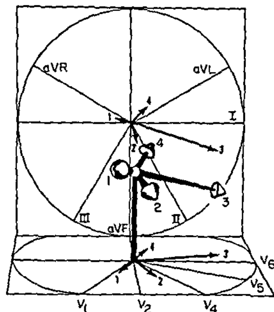


Fig 45 —The planar projections of the four normal instantaneous VA vectors Vector *1* represents initial septal activation vector *2* apicoanterior left ventricular wall depolarization vector *3* principally left ventricular depolarization and vector *4* terminal activation of basal portions of the left ventricle If the four QRS vectors described in Figure 44 are depicted as above their projections on the frontal plane lead axes and horizontal plane lead axes would cause leads I and V to register a small Q wave followed by a large R wave and leads aVR and V_1 to write a small initial R wave and then a deep S wave

septum and most of the lateral wall of the right ventricle have completed depolarization Except for a small posterobasal segment of the right ventricular walls the activation process is confined entirely to the left ventricle where it continues to spread through the thick lateral wall This gives rise to electrical forces of maximal strength which dominate the small right ventricular forces so completely that the resultant 0 04 second vector is almost identical with its left ventricular component Thus the 0 04 second vector is the largest of the VA vectors and is directed at almost a right angle to the long axis of the heart—i e to the left posteriorly and slightly inferiorly (approximately along the −5 or −10 axis of the frontal reference frame) (Fig 44 C) The appearance of this vector coincides with the inscription of the peak of the R wave in leads I and V_6 and the nadir of the S wave in V_1 (Fig 45)

0 06 second terminal or basal VA vector —The terminal vector is produced mainly by activation of the thick posterolateral and basal wall of the left ventricle Depolarization of the pulmonary conus also occurs at about this time but its over all contribution to the electrical field of the heart is ordinarily negligible The terminal vector is directed to the left and superiorly (approximately along the −40 axis in the frontal reference frame) and more posteriorly than the preceding vectors (Fig 44 D) and it projects smaller positive voltages on leads I and V_6 and smaller negative voltage on lead V_1 (Fig 45)

Although a mean cardiac spatial vector exists for each and every instant of the QRS interval for simplification only the four instantaneous VA vectors just described will be utilized hereafter to schematize ventricular activation In relating these spatial vectors to certain features of the electrocardiogram it should be kept in mind that the precordial lead deflections are largely determined by the projections of the cardiac vectors on the horizontal plane the limb lead deflections by the projections of the vectors on the frontal plane

Precordial transition of the QRS configuration —The normal precordial electrocardiogram is characterized by a progressive right *to left* increase in the relative amplitude of the R waves and a decrease in depth of the S waves The explanation offered for this finding by proponents of the unipolar or semidirect *lead* concept of electrocardiography is summarized later in this section As previously indicated the growing predominance *of left ventricular forces during the first* half of the QRS interval tends to be paralleled by an increasing magnitude and more leftward and posterior *orientation of each successive instantaneous vector* This trend culminates in the appearance of the maximal mean instantaneous QRS vector (corresponding *to the* 0 04 second VA vector) after which subsequent instantaneous vectors tend to decrease in length In other words as the instantaneous vectors lengthen they also rotate toward the positive halves of the lead axes of left precordial leads and away from the negative halves of the axes of right precordial leads If the horizontal projections of all the instantaneous cardiac vectors were evident rather than just those of the four VA vectors described it would become apparent that for each precordial lead there is an instantaneous vector which *projects maximal positivity* on the lead For example the 0 01 or 0 02

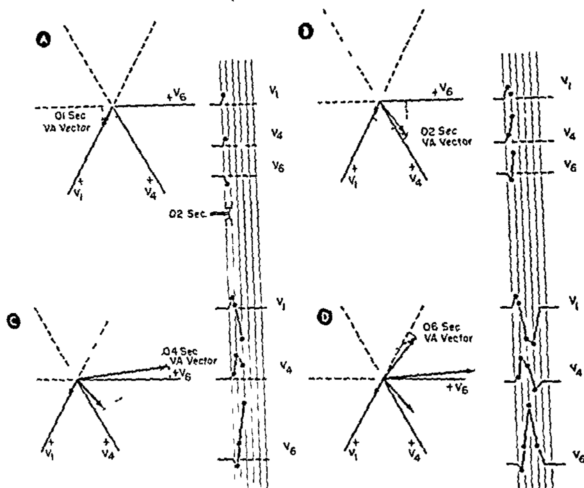


Fig 47 —A–D projections of the four *normal* instantaneous VA vectors on precordial leads V V and V_6 The voltages projected b
which in lead V h
second VA vector
in this lead coinci
the 0 04-second V
0 04 second after onset of ventricular activation

lead V_1 coincides with the appearance of the 0 01 second VA vector which is the *maximal* positive vector for the lead In contrast the peak of the R wave in V_6 coincides with the 0 04 second VA vector the maximal positive vector for lead V_6 It follows that the onset of the intrinsicoid deflection occurs later in lead V_6 than in V_1 but this merely indicates that the maximal positive instantaneous vector for V_6 appears later in the QRS interval than the corresponding vector for V_1 Moreover the appearance of the maximal positive vector for a given lead cannot be considered indicative of the completion of depolarization in the heart muscle facing the lead and this holds true whether ventricular activation takes place in a normal or abnormal manner One reason for this is that radial spread of the activation wave through the ventricular wall a prerequisite of Wilson's concept of the intrinsicoid deflection may not be characteristic of the abnormal or norm l hea

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occur

circumstance in itself invalidates Wilson's concept of the intrinsicoid deflection In actuality the authors have not found measurement of the ventricular activation time to be of much value in the clinical interpretation of electrocardiograms

lead to the left of leads V_3 and V_4 it was reasoned that clockwise rotation of the heart on its longitudinal axis must have occurred to bring the septum into this position Conversely counterclockwise rotation was implicated when the transitional lead was shifted to the right of leads V_3 and V_4 While there are many theoretical and practical objections to the electrocardiographic diagnosis of cardiac rotation it is quite conceivable that in an occasional instance anatomic rotation of the heart can be largely responsible for the pattern of clockwise or counterclockwise rotation* in the precordial leads

As already indicated in Chapter 3 it has been demonstrated that the lead recording the transitional QRS deflection is not necessarily the lead overlying the septum, but rather the lead whose axis is perpendicular to the horizontal mean QRS vector If for example the instantaneous VA vectors were added vectorially the horizontal mean VA vector expressing the average direction and magnitude of the instantaneous vectors would be found to lie along the −15 axis Since the lead axis of V_3 is situated at about +75 in the horizontal reference frame the mean VA vector is perpendicular to this lead axis and lead V_3 would record the transitional QRS complex Therefore in the precordial electrocardiogram the location of the lead registering the transitional QRS complex depends on the orientation of the horizontal mean QRS vector and this vector in turn ordinarily approximates the orientation of the maximal instantaneous QRS vector in the horizontal plane

Intrinsicoid deflection —If the exploring electrode of a unipolar lead is applied directly to the epicardial surface of the heart to form a direct lead the lead records an R wave as the ventricular activation wave moves toward the electrode At the exact moment that the epicardial muscle underlying the electrode becomes depolarized the activation potentials disappear and an abrupt downstroke is inscribed extending from the peak of the R wave to the base line or to the nadir of the S wave if present This is called the *intrinsic deflection* If muscle in contact with the electrode is the last to be depolarized then the intrinsic deflection ends at the base line and *forms the final* limb of the QRS complex in the lead However if the underlying muscle is not the last to be activated the intrinsic deflection terminates at the nadir of the following S wave Wilson considered the interval extending from the onset of the QRS complex to the onset of the intrinsic deflection in a direct lead to be related both to the thickness of muscle between the exploring electrode and the ventricular cavity and to the speed of conduction of the activation impulse through this muscle Thus onset of the intrinsic deflection would be expected to occur later in the QRS interval in leads recorded over the thick left ventricular wall than in leads overlying the right ventricle Wilson and his associates believed with some reservations that the so called *intrinsicoid deflection* in the indirect unipolar precordial leads used in clinical electrocardiography corresponded to the intrinsic deflection of the direct epicardial leads (Fig 46 A) The interval from onset of the QRS to onset of the intrinsicoid deflection (sometimes designated the *ventricular activation time* or *pre intrinsicoid deflection time*) was considered normal if it did not exceed a maximum of 0 035 second in precordial leads facing the epicardial surface of the right ventricle and 0 055 second in leads overlying the epicardial surface of the left ventricle Seemingly Wilson s concept of the intrinsicoid deflection was justified by the observation of the delayed onset of the intrinsicoid deflection in ventricular hypertrophy and intraventricular conduction disturbances

Since the intrinsicoid deflection concept outlined above is based on the semidirect lead hypothesis its validity for surface leads such as those utilized in clinical electrocardiography is open to serious question According to the equivalent dipole or vector concept of surface lead electrocardiography the intrinsicoid deflection in an indirect lead is produced by the instantaneous QRS vector which projects maximal positivity on the lead and is not related directly to the state of activation of the muscle nearest the electrode (Figs 46 A and 47) This concept can be simply illustrated by considering the 0 01 0 02 0 04 and 0 06 second VA vectors The times of onset of the intrinsicoid deflection in leads V_1 and V_6 are measured along the base line of the electrocardiogram from the beginning of the QRS interval to the point demarcated on the base line by a perpendicular dropped from the peak of the R wave in each lead In this hypothetical example the peak of the R wave in

*Hereafter in this text "clockwise rotation and counterclockwise rotation will be enclosed by quotation marks only when these terms refer to the so-designated electrocardiographic patterns By this means the authors hope to convey to the reader their doubts concerning the relationship between anatomic and electrocardiographic rotation

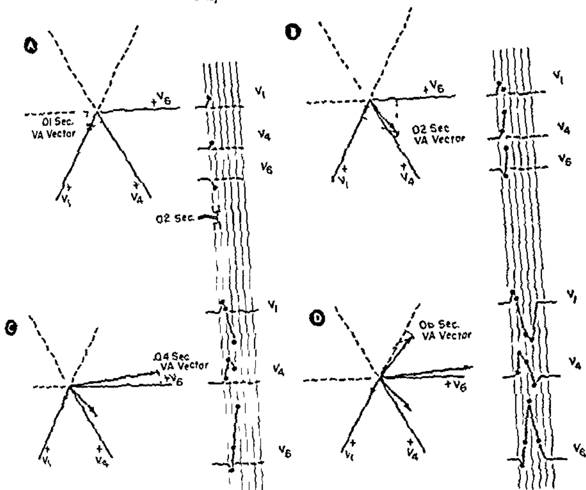


Fig 47 —A–D projections of the four normal instantaneous VA vectors on precordial leads V V and V The voltages projected by these vectors on the lead axes of the above le d ele ed — which in le d V … onset of ventricular activation

lead V_1 coincides with the appearance of the 0 01 second VA vector which is the maximal positive vector for the lead. In contrast the peak of the R wave in V_6 coincides with the 0 04-second VA vector the …

… ve instantaneous vector for V_6 appears later in the QRS interval than the corresponding vector for V_1 Moreover the appearance of the maximal positive vector for a given lead cannot be considered indicative of the completion of depolarization in the heart muscle facing the lead and this holds true whether ventricular activation takes place in a normal or abnormal manner One reason for this is that radial spread of the activation wave through the ventricular wall a prerequisite of Wilson's concept of the intrinsicoid deflection may not be characteristic of the abnormal or normal heart Tangential spread of excitation certainly occurs in the hypertrophied heart and may also occur in the normal heart (Fig 40 B and C) This circumstance in itself invalidates Wilson's concept of the intrinsicoid deflection In actuality the authors have not found measurement of the ventricular activation time to be of much value in the clinical interpretation of electrocardiograms

ELECTRICAL HEART POSITION

The relationship existing between the QRS configuration and the anatomic position of the heart in the chest has been recognized for many years. With the advent of unipolar electrocardiography, Wilson and his associates devised and later authors further developed a method by which five different *electrical* heart positions (the qualification electrical is deserving of emphasis) could be defined according to the particular combination of QRS complexes found in the unipolar limb and precordial leads. It is important to stress that Wilson regarded these electrocardiographic positions of the heart as being only generally *related to the anatomic heart position and not necessarily* dependent on it. The belief that a unipolar electrode records a QRS complex whose contour is relatively specific for the region of the heart facing the electrode was only one of the many applications of the semidirect lead hypothesis. Conversely, it was reasoned that if a QRS complex recorded by a given lead displays a configuration identified with a certain region of the heart, then the lead may be assumed to face this area. According to this concept, which has also been termed the *unipolar method* for electrocardiographic interpretation, potential variations of the *anterolateral epicardial surface of the left ventricle* are transmitted to the left precordium (leads V_5 and V_6 therefore recording qR complexes) and those of the epicardial surface of the right ventricle are transmitted to the right precordium (leads V_1 and V_2 *registering rS deflections*). *The right arm* (lead aVR) is thought usually to face the ventricular cavities and registers their negative potential variations as predominantly downward deflections (Qr, QS, rS or rSr′). The electrocardiographic positions of the heart and hence the configuration of the QRS complexes in aVL and aVF are thought to be expressions of rotation of the heart primarily on its longitudinal axis.

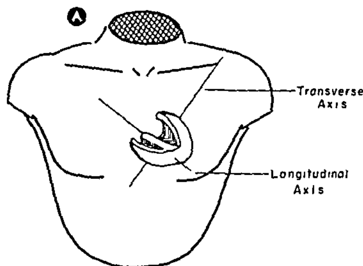
Electrical Axes of Rotation

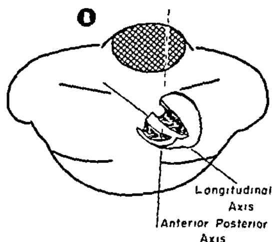
The axes of the heart have been defined as the *longitudinal*, *the anteroposterior and the transverse* axes.

Longitudinal axis.—This axis is a line connecting the apex with the midpoint of the ventricles (Fig. 48 A and B). Rotation on this axis is described as if the heart were visualized in an apex to base direction, clockwise rotation causing the right ventricle to move superiorly and counterclockwise rotation moving the left ventricle superiorly.

Anteroposterior axis.—This is a horizontal line running through the center of the heart in an anteroposterior direction (Fig. 48 B). Rotation of the heart *on this axis causes its apex to face either* the left shoulder (horizontal) or the left hip (vertical).

Transverse axis.—This is a line in the frontal plane perpendicular to and bisecting the longitudinal axis midway between apex and base of the heart (Fig. 48





axes

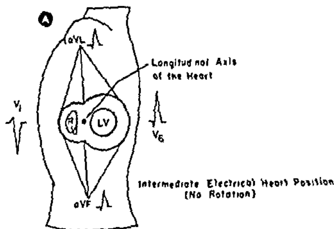
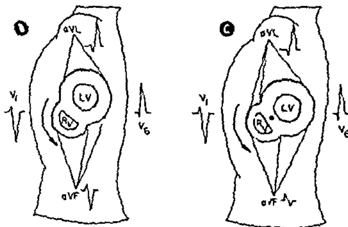
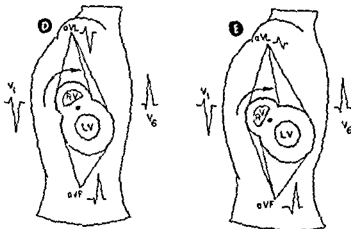


Fig 49 —Illustrating the genesis of the QRS patterns in the electrocardiographic heart positions in terms of the semidirect lead or unipolar concept According to this concept a lead facing only the right ventricle registers an rS complex one facing only the left ventricle a qR complex. Leads facing portions of both ventricles record QRS complexes which are intermediate in configuration between the rS and the qR complexes The heart is depicted in cross section through the ventricles as viewed through the left sagittal surface of the body—i e in an apex to-base direction. The longitudinal axis of the heart is seen end-on as a point lying in the interventricular septum Lead V is depicted as a posterior lead since it faces the posterolateral aspect of the left ventricle almost directly across the chest from lead V_1 (After Barker J M)





A) Rotation on this axis swings the apex of the heart forward or backward

Electrocardiographic Heart Positions

The following electrocardiographic heart positions are described in terms of the unipolar or semidirect lead concept (Fig 49)

Intermediate heart position —No rotation of the heart is considered to exist in the intermediate heart position (Fig 49 A) the right ventricle being anteriorly placed and the left ventricle posteriorly placed Leads aVL and aVF subtend equal angles with the left ventricular surface whose potential variations dominate those from the right ventricle Consequently the QRS deflections recorded in these two leads tend to resemble those registered in leads from the left side of the precordium (V_5 and V_6)

Horizontal heart position —Counterclockwise rotation of the heart on its longitudinal axis swings the left ventricle above the right ventricle which comes to occupy more of the diaphragmatic surface of the heart (Fig 49 B) Lead aVL therefore records potential variations conducted to it from the epicardial surface of the left ventricle so that its QRS deflections are similar to those recorded in leads V_5 and V_6 Lead aVF records electrical potentials from the right ventricle and exhibits QRS complexes resembling those in leads V_1 and V_2

Semihorizontal heart position —Less marked counterclockwise rotation results in lead aVL facing the left ventricle as above but lead aVF records potentials of about equal magnitude from those portions of the right and left ventricles resting on the diaphragm (Fig 49 C) Accordingly lead aVL registers complexes resembling those in leads V_5 and V_6 while lead aVF records very small complexes not resembling those of either side of the precordium

Vertical heart position —Clockwise rotation swings the right ventricle above the left ventricle which now comes to occupy more of the diaphragmatic surface of the heart (Fig 49 D) Lead aVL which faces the right ventricle records complexes resembling those in leads V_1 and V while lead aVF records QRS de

*When the source of electrical potential is a charged surface the potential at a field point (*P*) is proportional to the solid angle subtended by a cone drawn from *P* to include all surface dipoles on a sphere of unit radius about the point

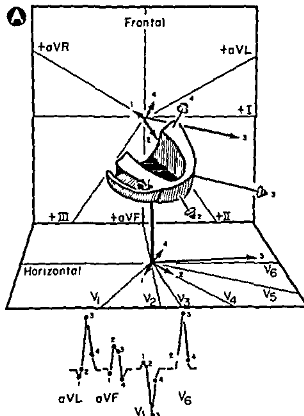
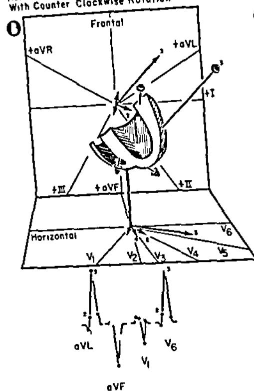
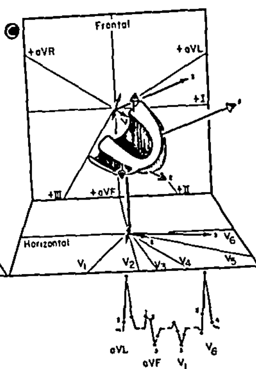


Fig 50 —Illustrating the genesis of the QRS patterns in the electrocardiographic heart positions in terms of the resultant vector concept In Figure 49 the QRS deflections in leads aVL and aVF and in leads V and V were explained in terms of the semidirect lead concept and were related to electrical heart position In the present [illegible] ds are re [illegible] of the [illegible] rt from which these vectors emanate in A through E has been depicted as if rotated in the same direction as the VA vectors This was done to indicate more clearly the direction of rotation of the vectors and should not be thought to imply that the heart itself actually shows such marked rotational changes

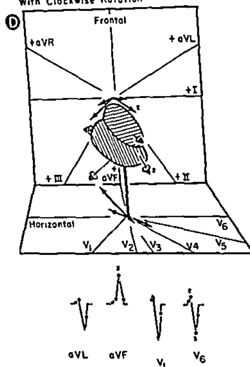
Horizontal Electrical Heart Position With Counter Clockwise Rotation



Semi Horizontal Electrical Heart Position



Vertical Electrical Heart Position With Clockwise Rotation



Semi Vertical Electrical Heart Position

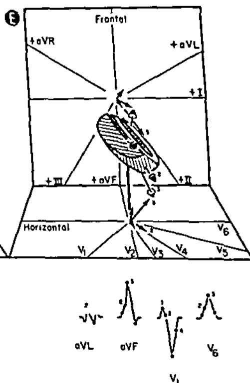


Fig 50 —Legend on facing page

flections similar to those in precordial leads V_5 and V_6

Semivertical heart position —With less marked clockwise rotation lead aVL faces portions of both left and right ventricles (Fig 49 E) Thus lead aVF records ventricular complexes resembling those in V_5 and V_6 while aVL registers deflections which are small and nondescript

Indeterminate position —There is no apparent relationship between the QRS complexes of the limb leads and those of the precordial leads

In terms of the equivalent dipole or vector concept the relationship between the configuration of the QRS complex in leads aVL aVF V_1 and V_6 can be *explained by varying degrees of rotation of the instantaneous vectors which may or may not be due to* anatomic rotation of the heart (Fig 50) Thus the 0 02 second VA vector approximating the long axis of the interventricular septum can serve as the axis around which the other instantaneous vectors can be rotated in a clockwise or counterclockwise direction It will be found that the projections of the four instantaneous VA vectors on the lead axes of the frontal and horizontal reference frames reproduce the electrocardiographic features described for the various electrical heart positions However, rotation of the instantaneous vectors does not necessarily mean that the heart itself has rotated as will be explained in Chapter 8 on ventricular hypertrophy

GENERAL CHARACTERISTICS OF THE NORMAL SCALAR ELECTROCARDIOGRAM

	EXTREMITY LEADS	PRECORDIAL LEADS
	P WAVE	
1 Direction	*In normal electrocardiograms the mean P spatial vector is situated to the left inferiorly and* somewhat anteriorly	
	The frontal mean P vector (A P) lies approximately *along the positive half of the lead* axis of lead II (+60) Therefore upright P waves are projected on leads I II and aVF inverted P waves on lead aVR Inasmuch as the frontal mean P vector lies more or less perpendicular to the lead axes of leads III and aVL minor variations in the position of the vector can completely reverse the direction of the P waves recorded in these leads Thus leads III and aVL can normally display upright inverted or diphasic P waves	The horizontal mean P vector usually is oriented *slightly to the right of the positive half* of the lead axis of lead V_1 (+30) or less frequently slightly to the left of this point Thus in the normal electrocardiogram precordial leads V through V_6 register upright P waves while lead V_1 records a low upright P wave if the mean P vector lies to the right of +30 an inverted P wave if the vector lies to the left of +30 and a diphasic P wave (+ −) if the mean P vector is oriented along the *+30* axis perpendicular to the lead axis of V_1
2 Amplitude	The amplitude of the P wave is not usually [illegible]	[illegible] interpretation of an
	Lead II generally records a taller P wave than leads I and III but in none of these leads does the normal P wave in children or adults equal or exceed 3 mm with only rare exceptions	In children and adults the amplitude of the normal P wave in lead V does not exceed 2 5 mm with only rare exceptions
3 Duration	The maximum normal duration of the P waves measured in *the bipolar limb leads is* 0 10 second P waves of 0 12 second duration [illegible]	
	broad P waves	

4 Comments

are recorded properly or not

4 Lead aVF

While the

ing in the atr

sion of retrog

right rarely

hypertrophy

observed in d

and occasional exceptions

P–R Interval

The P–R interval is measured from the beginning of the P wave to the onset of the first component of the QRS deflection The lead selected for measuring the P–R interval should be the bipolar limb lead with the longest P–R interval

Comments

percentage of normal subjects the P–R

the same

as much as 0 01 second or more

QRS Complex

1 Direction

In normal adults the mean QRS spatial vector is directed to the left inferiorly and slightly posteriorly (Fig 51)

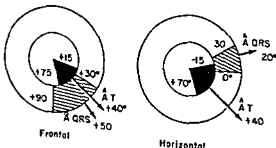


Fig 51 —The range of variation and average orientation of sÂ QRS and sÂ T in the frontal plane and horizontal plane calculated from the electrocardiograms of normal subjects

The frontal mean QRS vector is usually located between +30 and +90 and tends on the average to lie along the +50 axis Thus the main component of the QRS deflection is upright in leads I and II and is downwardly directed in lead aVR Leads III aVL, and aVF

The horizontal mean QRS vector is normally oriented between 0 and −30 its average position approximating −15

ally

recor

may normally record diphasic resultantly positive or resultantly negative QRS deflections The main component of the QRS should be upright in at least two of the three bipolar limb leads for any position of the frontal mean QRS vector between −30 and +150 In occasional normal obese adults the frontal mean QRS vector may be situated above +30 (left axis deviation) while in occasional tall slender adults and in children the mean frontal QRS vector may be situated to the right of +90 (right axis deviation)

and leads to the left recording *resultantly upright* qR or qRs deflections In normal subjects with the precordial lead pattern of *clockwise* rotation the transitional RS deflection may be recorded by a lead farther to the left (V) while counterclockwise rotation shifts the transitional lead to the right of V_2

---

2 Duration

In adults the QRS interval or width varies between 0 06 and 0 10 second in the limb leads In general the QRS duration tends to vary *inversely* with the heart rate and directly *with the age of the patient Thus in children* under 14 years of age the upper limit of normal for the QRS duration is 0 09 second in children under 5 years it is 0 08 second About 3% of normal adult subjects may have QRS deflections wider than 0 10 second

The QRS interval or duration is not ordinarily measured in the precordial leads This is

that the QRS interval can show such wide variation in the precordial leads of serial electrocardiograms recorded from the same individual

---

3 Amplitude

The criteria for high QRS voltage in the *limb leads are discussed in later chapters* (see Chapter 9 on Left Ventricular Hypertrophy and Chapter 10 on Right Ventricular Hypertrophy) in conjunction with the diagnostic criteria of ventricular hypertrophy In brief a

mm can be considered abnormally high voltage *High QRS voltage is usually not diagnosed from the bipolar limb leads*

Abnormally low QRS voltage exists if none of the three bipolar limb leads displays upwardly or downwardly directed QRS deflections exceeding 5 mm in amplitude (using the top or bottom of the baseline as the case may be for reference)

The criteria for *high QRS voltage in the precordial leads are somewhat complicated* and are best deferred for discussion along with the criteria for the diagnosis of ventricular hypertrophy Low QRS voltage is present in the precordial leads if the amplitude of the largest QRS deflection is 8 mm or less

---

4 Comments

Low QRS voltage can be observed in either the limb leads or the precordial leads of normal subjects and is usually due to the fact that the cardiac forces are oriented more or less perpendicular to the frontal plane or as the case may be the horizontal plane On the other hand when the QRS voltage is low in both limb and precordial leads then it can generally be inferred either that the magnitude of the cardiac forces is less than normal or that the cardiac forces are not transmitted to the surface electrode as well as normally whether because of decreased conductivity of the lungs as the result of emphysema or pleural or pericardial effusion or because of anasarca myxedema etc

A *electrode is moved from right to left across the precordium* the R/S amplitude the R nes be the R en the waves in leads V and V since the exploring electrode of leads V_3 and V_4 are closer to the chest electrodes and the heart electrocardiograms not infrequently show so gradual o may remain essentially the same in two or more eft decrease in the R/S ratio involving one or more his finding frequently indicates anterior myocardial ight ventricular hypertrophy

NORMAL Q WAVE

1 Duration

The presence of Q waves of 0.04-second duration in any of the precordial leads other than V_1 should raise the question of possible myocardial infarction although this abnormality is sometimes observed in other conditions such as left ventricular hypertrophy and chronic pulmonary heart disease. QS deflections can be present in lead V_1 in normal electrocardiograms but are probably not so common a finding as previously thought.

2 Depth

The depth of the normal Q wave in any of the twelve routine electrocardiographic leads other than leads aVR, aVL, and V_1 rarely equals and normally never exceeds 25% of the amplitude of the following R wave. The criteria of normal and abnormal Q wave amplitude in lead aVL are applicable only in conjunction with other criteria (see Chapter 19).

VENTRICULAR ACTIVATION TIME

(Preintrinsicoid deflection time or time of onset of intrinsicoid deflection)

The intrinsicoid deflection begins at the peak of the R wave ...

... the intrinsicoid deflection ... conduction disturbances and ventricular hypertrophy.

Q–T INTERVAL

The Q–T interval extends from the beginning of the ...

Bazett's formula for ... satisfactory

$$Q\text{–}T\ \text{calc}\ \text{or}\ Q\text{–}T_c = \frac{Q\text{–}T\ (\text{measured})}{\sqrt{R\text{–}R\ \text{interval (sec)}}}$$

A value for the Q–T calculated exceeding 0.425 second can be considered a prolonged Q–T for any age or sex but may or may not represent an abnormality.

RS–T OR S–T INTERVAL AND SEGMENT

The duration of the S–T ... ginning of the T wave (the ... even reasonable certainty) ... Q–T interval.

The S–T segment ... althou...

reference level for measuring S–T segment displacement is the T–P segment but in the presence of sinus tachycardia the P–R segment may offer a more accurate base line for comparison When S–T segment elevation occurs as a normal variation the S–T segment is often upwardly concave and is followed b... is less likely to be a normal variant ... tion of relatively low amplitude C... S wave of large area elevated S– ... since they probably do not signify subepicardial myocardial injury S–T segment elevation occurring as a normal variation tends to appear more commonly in leads V_1 and V_2 than in any of the other leads of the routine electrocardiogram

T WAVE

1 Duration	0 10–0 25 second Just as in the case of the S–T interval the duration of the T wave is generally not measured specifically but is included in the Q–T interval	
2 Direction	The T waves are normally upright in leads I and II b... in lead ... record ... not T ... ity although occasional exceptions to the rule are noted	Usually the T waves are upright in precordial leads V_2 through V_6 but normal adults under 30 years of age may show inverted T waves in leads V_1 through V_3 and sometimes in V... However in adults over 30 T wave inversion is usually confined to lead V_1 Inverted T waves in leads V_5 and V_6 have the same significance as in lead I although in none of these leads does an inverted T wave per se necessarily constitute an abnormality
3 Amplitude	Although it is stated that the amplitude of the normal T wave in the limb leads only rarely exceeds 6 mm there is rather marked individual variation In fact amplitude criteria for T wave normality or abnormality have not been established	

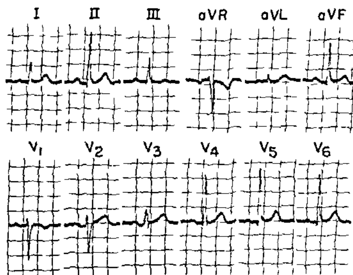


Fig 52 –Normal electrocardiogram In the frontal plane Â P = +60° Â QRS = +60 and Â T = +45 In the horizontal plane Â QRS = −10 and Â T = +20 The orientation of sÂ QRS and sÂ T in the horizontal plane can only be determined if one of the routine precordial leads records the transition from resultantly upright QRS and T deflections to resultantly inverted deflections or vice versa In the above electrocardiogram the transitional lead for QRS is situated between leads V and V_3 Since Â QRS must be perpendicular to the transitional lead the orientation of the vector is approximately −10 to −15 The transition lead for T is located between leads V and V upright T waves being recorded to the left and inverted T waves to the right Again since $\hat{A}_1$ T must be

relatively narrow

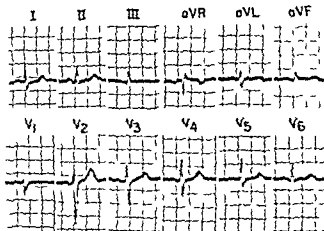


Fig 53 – Normal electrocardiogram In the frontal plane Â QRS is situated at about +60 and Â T is located at +30 In the horizontal plane the Â QRS is at −25 and Â T is at +35 Although this electrocardiogram shows semivertical heart position as a rule electrical heart position will not appear in the interpretation of any subsequent electrocardiograms in this text

4 Comments

Ventricular Repolarization, Ventricular Gradient and Spatial QRS-T Angle

VENTRICULAR REPOLARIZATION

The S–T Segment and T Wave

THE ISOELECTRIC SEGMENT which is written immediately following termination of the QRS deflection and extends into the T wave is called the *RS–T segment* or *S–T segment* and the corresponding time interval *is referred to as the S–T interval* Electrophysically the S–T segment is the earliest part of the T wave Normally recovery begins in some regions of the heart before depolarization is completed in other areas However these early repolarization forces which are produced during the S–T interval do not normally reach sufficient magnitude to displace the S–T segment from the isoelectric base line In the occasional *normal exceptions to this rule minor degrees* of S–T segment elevation (usually not exceeding 2 5 mm) are observed most commonly in leads V_1 to V_3 The predilection of this normal variation for leads V_1 *through* V_3 *is probably explained by* one or more of the following three factors

1 The closer proximity of the exploring chest electrodes of leads V_1 to V_3 to the heart would tend to magnify all the electrical forces produced by the heart and among these the early repolarization forces This may actually represent one of the few instances in which the precordial leads respond in part to *proximity* or *localized potentials*

2 In the discussion of the ventricular gradient later in this chapter an important relationship is explained which is pertinent to the present subject namely that the larger the resultant area of the QRS deflection the greater will be the tendency for the S–T segment and T wave to be displaced in a direction opposite to that of the QRS deflection Thus since leads V_1 through V normally record QRS deflections with relatively large terminal S waves the S–T segments in these leads are more likely to be elevated than the S–T segments in other leads

3 Occasional instances of apparent S–T segment elevation appearing in lead V_1 or less frequently in lead V may we believe actually represent a small terminal R which merges indistinguishably with the S–T segment In such cases we have observed rSr′ deflections in leads V_1 and/or V of electrocardiograms recorded prior or subsequent to the tracing showing the apparent S–T segment elevation Vector cardiograms obtained in a few cases have not shown an S–T vector but instead have displayed terminal QRS vectors which were directed slightly to the right

attributed to late depolarization in the region of the pulmonary conus

As will be recalled a vector representing elementary repolarization forces points away from the direction in which the repolarization process is spreading

the positive charges of the dipoles along the wave front precede the negative charges It so happens *that in depolarization the orientation of electrical* positivity and thus the direction the vector points coincides with the direction of physiological activity

In the isolated cell or muscle strip repolarization and depolarization begin at the same point on the cell surface and proceed in the same direction However the intact heart muscle cell in situ and in a more

of depolarization The fact that unlike the recovery process in the isolated cell repolarization of the muscle of the ventricular wall is initiated in the layer of muscle last activated has been attributed to subendocardial recovery delay resulting from the great pressure applied to the inner ventricular wall during dynamic cardiac systole The delayed onset of subendocardial repolarization permits epicardial muscle to recover first even though it was the last activated and, as a consequence the repolarization wave moves toward the endocardial surface of the heart Since the repolarization wave advances toward depolarized muscle and leaves polarized muscle in its wake the instantaneous repolarization forces and representative mean instantaneous T vectors point in a direction the opposite of that in which repolarization is spreading Parenthetically one might add that atrial myocardium unlike ventricular muscle behaves in the same manner as the isolated muscle strip in that repolarization begins in the same region and proceeds in the same direction as depolarization For this reason the P wave of atrial depolarization and the atrial T wave (Ta wave) are oppositely directed

The average direction and magnitude of all mean instantaneous T spatial vectors which produce the electrocardiographic T wave can be represented by a single vector the mean T spatial vector (sÂT) Usually the mean T spatial vector of normal subjects points to the left inferiorly and either anteriorly or slightly posteriorly It is more likely to have a vertically inferior orientation in younger persons and a horizontal and leftward orientation in older subjects The most constant feature of the normal mean T spatial vector is its approximately parallel orientation with respect to the mean QRS spatial vector The spatial QRS–T angle subtended by the two mean spatial vectors which is described later in this chapter rarely exceeds 50–60° in the absence of detectable heart disease This accounts for the fact that the T waves are normally upright in leads I V_5 and V_6 since these leads record resultantly upright QRS deflections in most instances

The U Wave

The U wave is usually a minor upright wave which follows the T wave or is fused with it The exact cause of this deflection is not known but there is reason to believe that it is produced by potentials elicited by the stretching of ventricular muscle during the period of rapid blood inflow into the ventricles A negative U wave is never present in normal subjects in leads I and II h ... vascul ... Appar ... is obse ... u characteristically in many of the electrocardiographic leads when the serum potassium concentration is quite low This is discussed more fully in a later chapter

VENTRICULAR GRADIENT AND SPATIAL QRS–T ANGLE

In the n ... fundamen ... tion as a n ... the depol ... wave front in that the negative charges of the dipoles along the repolarization wave front precede the positive charges the reverse being true in the case of depolarization Useful as this simplification may have been repolarization of heart muscle is such a protracted process compared to depolarization that except at the beginning and end recovery is occurring at the same time throughout most of the muscle It is evident from this that the electrical effects of repolarization of some portions of heart muscle or in the case of a single muscle cell of some areas of the cell membrane tend to offset the effects of repolarization occurring simultaneously at other sites In other words there is *opposition of effects* in repolarization This is not the case in depolarization for the latter process can be visualized as a wave front of dipoles involving at any instant only a small strip of cell membrane or of muscle tissue From a practical standpoint however only the potential variations at the endocardial and epicardial surfaces of a heart muscle cell need be considered since the electrical effects of depolarization and repolarization of a single myocardial cell—or for that matter the entire thickness of ventricular muscle—are equal to the algebraic sum of the electrical effects of these processes at the endocardial and epicardial surfaces of the cell or ventricular wall For this reason it is a permissible simplification to imagine the ventricular muscle to be a single muscle fiber extending from the endocardium to the epicardium of the heart

That only the surface effects of depolarization and repolarization need be considered is generally accepted by most authorities in the field of electrocardiography This premise has been the basis of a number

Ventricular Repolarization, Ventricular Gradient and Spatial QRS-T Angle

VENTRICULAR REPOLARIZATION

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THE ISOELECTRIC SEGMENT which is written immediately following termination of the QRS deflection and extends into the T wave is called the *RS–T segment* or *S–T segment* and the corresponding time interval is referred to as the *S–T interval*. Electrophysically the S–T segment is the earliest part of the T wave. Normally recovery begins in some regions of the heart before depolarization is completed in other areas. However these early repolarization forces which are produced during the S–T interval do not normally reach sufficient magnitude to displace the S–T segment from the isoelectric base line. In the occasional normal exceptions to this rule minor degrees of S–T segment elevation (usually not exceeding 2.5 mm.) are observed most commonly in leads V_1 to V_3. The predilection of this normal variation for leads V_1 through V_3 is probably explained by one or more of the following three factors:

1. The closer proximity of the exploring chest electrodes of leads V_1 to V_3 to the heart would tend to magnify all the electrical forces produced by the heart and among these the early repolarization forces. This may actually represent one of the few instances in which the precordial leads respond in part to *proximity* or *localized potentials*.

2. In the discussion of the ventricular gradient later in this chapter an important relationship is explained which is pertinent to the present subject, namely that the larger the resultant area of the QRS deflection the greater will be the tendency for the S–T segment and T wave to be displaced in a direction opposite to that of the QRS deflection. Thus since leads V_1 through V_3 normally record QRS deflections with relatively large terminal S waves, the S–T segments in these leads are more likely to be elevated than the S–T segments in other leads.

3. Occasional instances of apparent S–T segment elevation appearing in lead V_1 or less frequently in lead V_2 may, we believe, actually represent a small terminal *R* which merges indistinguishably with the S–T segment. In such cases we have observed rSr′ deflections in leads V_1 and/or V_2 of electrocardiograms recorded prior or subsequent to the tracing showing the apparent S–T segment elevation. Vector cardiograms obtained in a few cases have not shown an S–T vector but instead have displayed terminal QRS vectors which were directed slightly to the right and posteriorly. The latter finding and the corresponding rSr′ deflection in leads V_1 of the electrocardiogram usually represent a normal variation and have been attributed to late depolarization in the region of the pulmonary conus.

As will be recalled, a vector representing elementary repolarization forces points away from the direction in which the repolarization process is spreading since the negative charges of the dipoles along the repolarization wave front precede the positive charges. This is to be contrasted with depolarization in which *the positive charges of the dipoles along the wave* front precede the negative charges. It so happens that in depolarization the orientation of electrical positivity and thus the direction the vector points coincides with the direction of physiological activity.

In the isolated cell or muscle strip repolarization and depolarization begin at the same point on the cell surface and proceed in the same direction. However, the intact heart muscle cell in situ and in a more general sense the ventricular myocardium as a whole undergo *repolarization in an epicardial to endocardial* direction, this being just the reverse of the direction

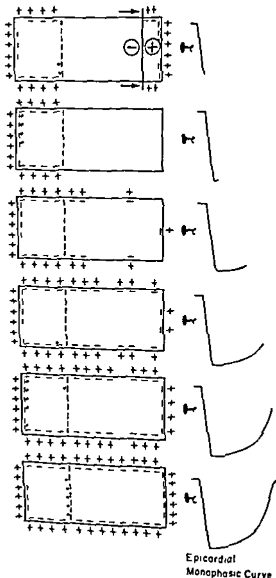
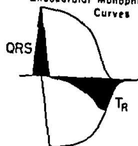


Fig 54 (*cont*) —In **B** the endocardial surface of the cell has been cooled Consequently the downwardly directed curve recorded at the epicardial surface represents solely potential variations at the latter surface the curve being designated the *epicardial monophasic curve* Because the same number of dipoles have been involved in depolarization and repolarization of the epicardial surface and of the endocardial surface and because of the uniform rate of recovery throughout the cell it follows that the endocardial and epicardial monophasic curves have the same configuration and enclose equal areas even though they are opposite in sign or direction

If the upright endocardial monophasic curve and downwardly directed epicardial curves described in **A** and **B** are plotted on the positive and negative sides of the same base line (as in **C**) with the endocardial curve slightly preceding the epicardial curve (since depolarization and repolarization of the cell are assumed to start first at its endocardial surface) the potential variations between the two cell surfaces during depolarization and repolarization can be obtained by algebraic addition of corresponding points on the two curves The values obtained plotted against time according to their voltage and sign yield an upright R wave and inverted T wave of equal area but opposite sign The T wave obtained when there is uniform [illegible]
o
F
A



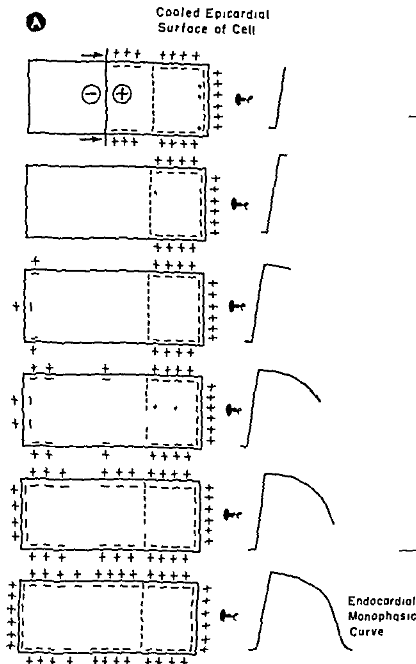


Fig 54 —The endocardial and epicardial monophasic curves recorded from a heart muscle cell and their relationship to the QRS deflection and regression T wave In A the cooled epicardial surface of the cell undergoes no

boundary of the cooled region which therefore retains its dipoles As a consequence the monophasic curve recorded by the electrode overlying the epicardial end of the cell reflects entirely potential variations at the endocardial surface of the cell and is called the *endocardial monophasic curve* Note that repolarization is occurring more or less simultaneously throughout the cell It begins at the endocardial surface and since the rate of recovery is uniform throughout the cell the endocardial region regains all of its dipoles sooner than regions of the cell nearer the epicardial surface Thus the repolarization process moves in an endocardial to-epicardial direction but does not produce a wave front (*continued*)

of classic studies by Wilson and his associates and by Bayley Ashman Gardberg Byer and others The results of these investigations can be summarized in the form of the following hypothetical experiment

Part I (Fig 54 A) —*If one first cools the epicardial surface of the cell and then stimulates the endocardial surface* the depolarization wave spreads from the site stimulated toward the epicardial end of the cell (*The direction of physiologic activity in the cell will be assumed in this experiment to parallel the axis of a unipolar lead whose exploring electrode lies in close proximity to the epicardial membrane surface*) However the activation wave is blocked at the boundary of the cooled region which therefore retains its membrane dipoles even though the rest of the cell surface has been discharged With discharge of the endocardial aspect of the cell the epicardial surface becomes intensely positive by comparison so that an abrupt upward deflection of the electrocardiographic base line occurs The summit of this curve persists until repolarization commences at the endocardial surface With the appearance of the first dipoles on the endocardial surface the relative positivity of the cooled *epicardial surface begins to decline* As recovery through the cell continues the number of dipoles on the endocardial surface increases progressively and as they approach in number those present during the resting phase the

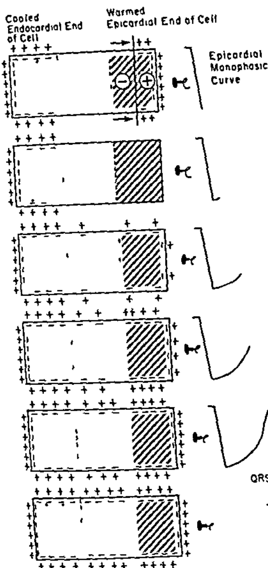


Fig 56 —The effect of warming the epicardial of the heart muscle cell on the recorded T

(which is the same as in Figures 54 and 55) ally QRS and T

nated G the ventricular gradient This symbol (G) represents the net electrical effect of the difference in the rates of endocardial and epicardial repolarization

of two waves the first the QRS which represents the net electrical effects of depolarization and is upright in this experiment and the second the T wave which represents the net electrical effects of repolarization and is in this instance downwardly directed When there is a uniform rate of recovery throughout the cell as is assumed in this phase of the hypothetical experiment, the T wave is equal in area but opposite in direction to the QRS deflection and is called the *regression T wave* (T_R) It follows from the definition of a regression T wave that A QRS + A $T_R = 0$ where A equals area This important relationship should be kept in mind during the remainder of the hypothetical experiment

Part III (Fig 56) —In this phase of the experiment the epicardial surface of the cell is warmed and the endocardial surface cooled Depolarization is blocked at the boundary of the cooled cell membrane the endocardial surface retains its dipoles and consequently discharge of the epicardial surface of the cell causes a sharp downstroke of the electrocardiogram tracing just as occurred in Part II However because the epicardial end of the cell has been warmed it recovers more quickly than previously The greater rapidity with which the epicardial surface of the cell regains its dipoles during repolarization is reflected in the more rapid return of the monophasic curve to the isoelectric base line the duration

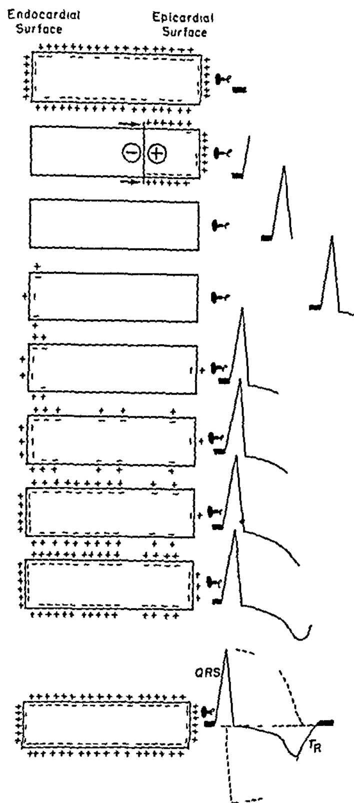
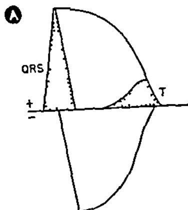


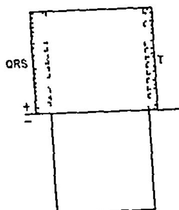
Fig 55 —The QRS complex and regression T wave recorded at the epicardial surface of a heart muscle cell in [illegible] form through [illegible] s [illegible] cardial electrode in this illustration where [illegible] cal surface has been cooled

electrocardiogram curve returns gradually to the isoelectric base line When repolarization is completed the endocardial and epicardial surfaces are equally charged and the curve reaches the isoelectric base line Inasmuch as there has been no change in *the number of dipoles at the epicardial surface of* the cell during this period the monophasic curve recorded must therefore represent the electrical effects of depolarization and repolarization of the endocardial end of the cell The duration of the endocardial monophasic curve is a function of the rate of recovery at the endocardial surface of the cell

Part II (Fig 54 B) —*If one next cools the endo*cardial end of the cell an electrode overlying the opposite end records an abrupt downward deflection at the time the epicardial surface is discharged At this point the epicardial surface of the cell becomes negatively charged relative to the cooled endocardial surface which has retained its dipoles for the reason previously cited With the reappearance of the first dipoles on the epicardial surface the electrocardiographic tracing begins its ascent to the base line As the dipoles increase in number the positivity of the endocardial surface with reference to the epicardial surface declines progressively and a rising curve is inscribed which reaches the isoelectric base line with completion of repolarization In this instance the monophasic curve obtained is exactly like that recorded when the epicardial surface was cooled except that in the latter case the curve was upright while the curve just recorded is downwardly directed *This negative monophasic curve represents the elec*trical effects of epicardial depolarization and repolarization since the degree of polarization of the endocardial surface of the cell did not change significantly during the activation and recovery processes

The two monophasic curves obtained in Parts I and II of the experiment are now plotted on the positive and negative sides of the same base line with the endocardial curve slightly preceding but overlapped terminally by the epicardial curve since the endocardial surface of the cell was the first to be *activated and the first to recover (Fig 54 C) Each* monophasic wave represents the depolarization and repolarization potentials existing at one or the other cell surface plotted against time The potential variations between the two cell surfaces can be determined *by algebraically adding pairs of* corresponding points on the monophasic curves The values obtained are plotted according to their voltage and sign and yield a diphasic deflection This derived deflection is identical with the deflection which would be recorded if neither cell surface were cooled (Fig 55) It consists





B

$\hat{G} + (\ \hat{A}T_R)\ \hat{A}T$

1) $-\hat{A}T_R$ $\hat{A}T$ $\hat{G}$ +

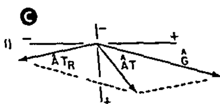
2) $-\hat{A}T_R$ $\hat{A}T$ + $\hat{A}T - \hat{A}T_R$ $\hat{G}$ $\hat{G}$

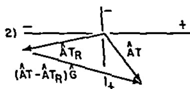
But, $\hat{A}T_R\ \ -\hat{A}QRS$

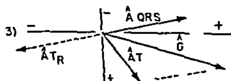
Therefore $\hat{A}T-(-\hat{A}QRS)\ \ \hat{G}$

or $\hat{A}T + \hat{A}QRS\ \ \hat{G}$

3) − $\hat{A}T$ + $\hat{A}$ QRS $\hat{A}$ QRS $\hat{A}T$ $\hat{G}$







... ecto Â QRS Â T and Â T In A are shown the ... me n lrs of monn

(B sec 3) The same relationships between Â QRS Â T and Â T are demonstrated in parts 1–3 of C in which the vectors do not lie along the axis of the electrocardiographic lead recording the monophasic curves

Fig 57 —The ventricular gradient and its relationship to the endocardial and epicardial monophasic curves In A *the two monophasic curves obtained when there is uniform rate of recovery throughout the cell have been added alge-*

directions In B the endocardial curve is the same
the endocardial curve and the shortened epicardial
difference in the areas enclosed by the epicardial
curve of A and the shortened epicardial curve (as represented by lines *A* to *G* of B equals the total area enclosed by the upright T wave and the regression T wave (represented by lines *A* to *G* extending between the two T waves lines which are equal in length to corresponding lines between the two superimposed monophasic curves) The sum of A T_R + A T is designated the gradient or G Therefore A T_R + A T = G

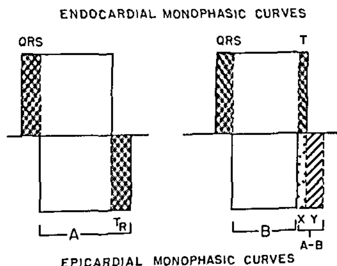


Fig 58 —The relationships described in Figure 57 *can be expressed more simply by substituting* rectangles as here for the monophasic curves Rectangle A is the epicardial curve yielding the

fore Area (A) − Area (B) = A (X) + A (Y) = A T + A T_R = G

of the curve being shortened to a corresponding degree. Since the endocardial surface of the cell has not been warmed, the rate of recovery there is the same as before, and so the endocardial monophasic curve remains unchanged from that recorded in Part II. As a result, the endocardial and epicardial monophasic curves are no longer equal in area and, when added algebraically, yield QRS and T waves of differing areas. Thus the sum of (Ā QRS) and (Ā T) does not equal zero but instead equals a measurable quantity (Ĝ), designated by Wilson the *ventricular gradient*. Clearly, the ventricular gradient represents the net electrical effect of the difference in the rates of repolarization at the endocardial and epicardial surfaces of the cell. To reduce the ventricular gradient concept to more tangible terms, it is defined in the following paragraph as a function of the monophasic curves obtained in Parts II and III of the hypothetical experiment.

If the epicardial monophasic curve from which the inverted regression T wave was derived and the shortened monophasic curve of the upright T wave last recorded are superimposed on each other, the difference in their areas, which will be found to equal the total area enclosed by the two T waves, is identical with the ventricular gradient (Figs 57 and 58). In other words, the ventricular gradient represents the repolarization potentials which, added to the regression T potentials, yield the potentials producing the upright T wave (Fig 59). If these electrical forces are depicted as vector quantities, vector addition of the regression T vector and ventricular gradient vector yields, as the resultant of the two component vectors, the recorded T vector. Theoretically it is possible to calculate the ventricular gradient by subtracting vectorially the regression T vector from the recorded T vector. In actuality, however, there is no way to record the regression T wave of heart muscle because the rate of repolarization is not uniform throughout the ventricular muscle. Nevertheless, since the QRS area and the regression T area are of equal size, although of opposite sign, the ventricular gradient can be determined by vector addition of Ā QRS and Ā T.

As stated earlier, the axis of derivation of the [illegible]

Consequently, in determining the ventricular gradient the vectors representing Ā QRS and Ā T must be laid off along the lead axis. In Parts I and II of this experiment the rate of repolarization was uniform throughout the cell, and the epicardial and endocardial monophasic curves obtained enclosed equal areas of opposite sign. Added algebraically, the two monophasic curves yield QRS and T deflections which are also equal in area but opposite in sign. If Ā QRS and Ā T are plotted along the positive half of the axis of derivation of the lead recorded, the resultant of the two vectors (which in this instance equals the sum of Ā QRS and Ā T laid off head to tail, since the vectors coincide exactly in direction) is *not zero*, as was previously the case, but instead is a third vector, the gradient vector Ĝ. This gradient vector has a specific magnitude and is directed away from the region in which the duration of the excited state is longest (i.e., recovers slowest) toward the region where it is shortest. In this phase of the experiment the gradient vector is found to be directed from the endocardial surface of the cell toward the epicardial surface.

Part IV (Fig 60).—In this portion of the hypothetical experiment, the muscle cell is stimulated at its epicardial surface, and so the direction in which the cell undergoes depolarization is the *reverse* of that described previously. Repolarization will again be assumed to occur at a uniform rate throughout the cell, so that the epicardial and endocardial monophasic curves recorded are equal in area but oppositely directed. Since depolarization and repolarization both commence first at the epicardial surface of the cell, the epicardial monophasic curve precedes the endo- [illegible]

[illegible] of equal area, whereas previously, when activation of the cell occurred in an endocardial-to-epicardial direction, an upright QRS and inverted T wave were obtained. It is evident that this reversal in the direction of the QRS and T deflections can be attributed entirely to the reversed direction of depolarization. Because there has been no change in the uniform duration of the excited state throughout the cell, one is not surprised to find on plotting Ā QRS and Ā T on the axis of derivation of the electrocardiographic lead that once again the ventricular gradient is zero.

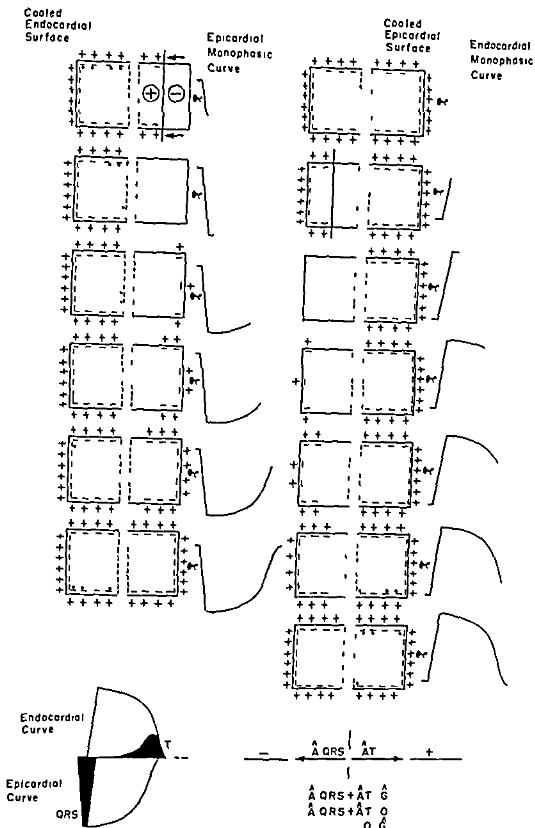


Fig 60 —The effect of reversal in the direction of cellular depolarization on the monophasic curves and on Â QRS and Â T_R Here the cell has been stimulated at its epicardial surface so that depolarization and repolarization of the

the endocardial surface Repolarization occurs at a uniform monophasic curves are equal but their time relationship to epicardial curve precedes the endocardial curve with the

No matter how great may be the discrepancy between the times of onset of depolarization and repolarization at the endocardial and epicardial surfaces of the cell as long as the rate of repolarization remains uniform through the cell the areas enclosed by the epicardial and endocardial monophasic curves remain equal and the ventricular gradient continues to be zero (Fig 61) Thus as onset of depolarization at one surface of the cell lags farther and farther behind the time of onset of depolarization at the other surface the derived QRS and T deflections enclose larger and larger areas of equal size but opposite sign and Â QRS + Â T = Ĝ = 0 One can demonstrate the preceding facts for himself in the following simple way

To obviate the necessity of having to add algebraically two monophasic curves the latter are represented as two rectangles of equal dimensions The "endocardial rectangle" is drawn above the isoelectric base line the "epicardial rectangle" below the base line (Fig 61) The left side of each rectangle cor-

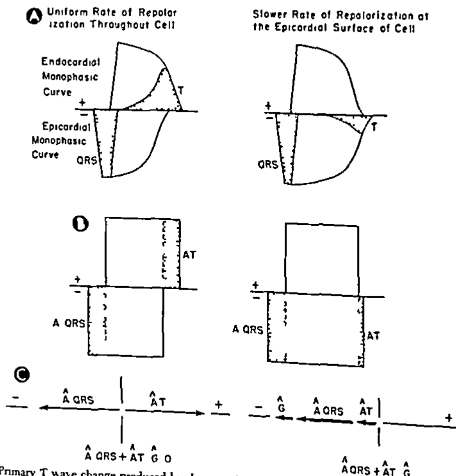
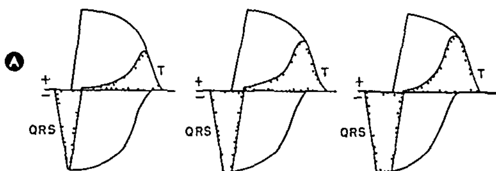
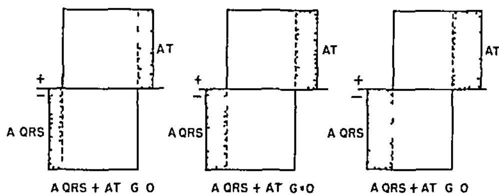
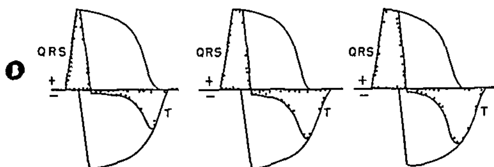


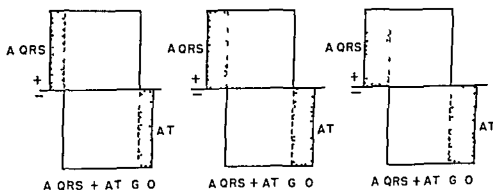
Fig 62 – Primary T wave change produced by slowing of ... cle cell. The monoph... addit... n of Â QRS ...

... addition of Â QRS and Â T derived from the ... clinical electrocardi...









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holds true

No matter how great may be the discrepancy between the times of onset of depolarization and repolarization at the endocardial and epicardial sur ... tion

remain equal and the ventricular gradient ... to be zero (Fig 61) Thus as onset of depolarization at one surface of the cell lags farther and farther behind the time of onset of depolarization at the other surface the derived QRS and T deflections enclose larger and larger areas of equal size but opposite sign and A QRS + A T = G = 0 One can demonstrate the preceding facts for himself in the following simple way

To obviate the necessity of having to add algebraically two monophasic curves the latter are represented as two rectangles of equal dimensions The "endocardial rectangle" is drawn above the isoelectric base line the "epicardial rectangle below the base line (Fig 61) The left side of each rectangle corresponds to the initial limb of the monophasic curve while the right side corresponds to the terminal limb of the curve When one rectangle precedes the other

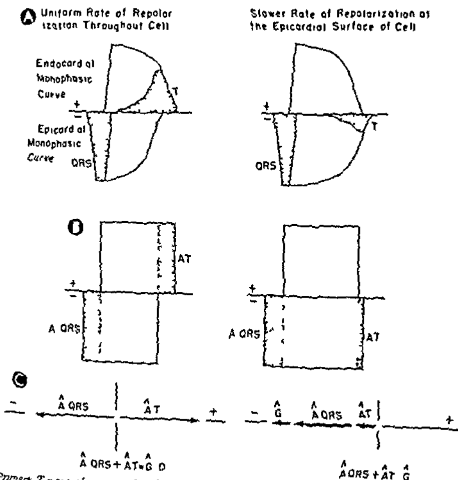


Fig 62 —*Primary* T wave change produced by slowing of ... cell ...

d A T derived *from the*

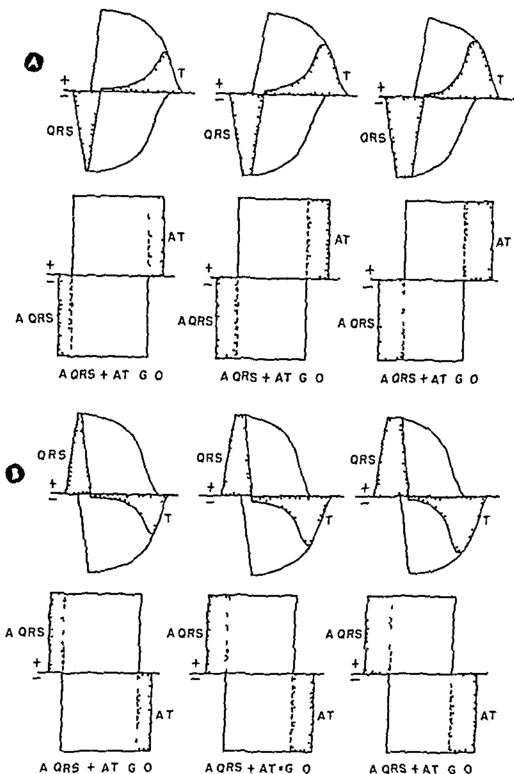


Fig 61 —Demonstration of the fact that no matter how marked the discrepancy between the times of onset of depolarization and repolarization at the two surfaces of the cell as long as the rate of repolarization remains uniform throughout the cell the areas enclosed by the epicardial and endocardial monophasic curves remain equal and the ventricular gradient continues to be zero In A epicardial depolarization precedes endocardial depolarization while in B the reverse holds true

the overlapping portion of the former (on the left) is analogous to the QRS deflection similarly if the right end of one rectangle extends beyond the other the overlapping area corresponds to the T wave

1 If the endocardial rectangle precedes (extends to the left of) the epicardial rectangle, the overlapping part of the former is situated above the base line and corresponds to an upright QRS deflection Since the dimensions of the two rectangles are equal it is obvious that the epicardial rectangle must necessarily overlap the right end of the endocardial rectangle to the same extent that its left end is overlapped by the latter Thus the overlapping portion of the epicardial rectangle corresponds to a T wave enclosing the same area as the QRS but directed oppositely Hence A QRS + AT = G = 0

2 If the endocardial rectangle continues to be advanced farther and farther ahead of (i e to the left of) the epicardial rectangle the overlapping portions of the two rectangles representing the QRS and T deflections increase in size but maintain their original directions and equal areas Thus the gradient is still zero just as in the above paragraph

3 To determine the effect on the gradient of a *complete reversal* in the time sequence of depolarization, initial stimulation of the epicardial surface of the cell will be simulated by moving the epicardial rectangle ahead of or to the left of the endocardial *rectangle In* this instance the left end of the epicardial rectangle situated below the base line overlaps the left end of the endocardial rectangle while the right *end of the latter overlaps to an equal extent* the right end of the epicardial rectangle The overlapping portions *of the* two rectangles are analogous to downwardly directed QRS and upright T deflections *of equal* area Here too A QRS + A T = G = 0 despite the fact that the directions of the QRS and T deflections are completely reversed compared to their directions in the preceding two paragraphs

The salient fact to emerge from the above discussion and one which is the very core of the ventricular gradient concept is this An [illegible] rer [illegible]
or [illegible]
a [illegible] [illegible] throughout the heart muscle is not accompanied by an alteration in the ventricular gradient.

Part 3 (Fig 62)—*In* continuation of the hypothetical experiment with *the equally hypothetical* heart muscle cell (which *extends from the* endocardial to the epicardial *surface of the ventricular* wall) the next step will be to cool the epicardial surface of the cell to a degree sufficient to delay recovery in the cooled region without blocking depolarization The stimulus is applied to the epicardial surface of the cell just as was done in *the preceding phase* of *the experiment* Since onset of depolarization occurs at the epicardial surface of the cell before its occurrence at the endocardial surface the direction of depolarization is the reverse of normal and the overlying lead electrode records a downwardly directed QS deflection However because the epicardial end of the cell has been cooled its rate of recovery is slowed relative to that of the endocardial surface which although the last activated reaccumulates its *dipoles* at a faster rate This reverses the general order of repolarization and so the epicardial surface and overlying lead electrode are at a relatively negative potential with respect to the endocardial end of the cell throughout the entire period of repolarization The T wave recorded is therefore directed downwardly (is inverted) like the QS deflection that preceded it Consequently if one were to record monophasic curves from the two surfaces of the cell one would note the following (1) Onset of the epicardial curve precedes onset of the endocardial curve (2) The epicardial curve is prolonged beyond the termination of the endocardial monophasic curve as the result of the slower rate of epicardial repolarization (3) Thus the endocardial curve is overlapped by the epicardial curve at both its beginning and end and obviously encloses a smaller area than the latter (4) The overlapping portions of the epicardial curve correspond on algebraic addition of the two curves to the downwardly directed QS and T deflections of differing areas which were previously recorded (5) The areas enclosed by the QS and T *deflections* can be represented by vectors which differ in length but point in the same direction If these vectors A QRS and A T are laid off along the negative half of the axis of derivation of the lead *recorded* and are then added vectorially the res [illegible]
but is a vecto [illegible]

Whatever [illegible]
the gradient [illegible]
ter what the [illegible] relationship between *the* epicardial and endocardial monophasic curves may be *provided* that the *areas enclosed* by the two curves maintain their same relative proportions (Fig 63) In other words the *presence* of a gradient initially is indicative not of the fact that the direction of depolarization is reversed but of the fact that there is a difference in *the rate of recovery* or in the duration of the excited state at *the endocardial* and epicardial surfaces of the cell As long as there is *no further change*

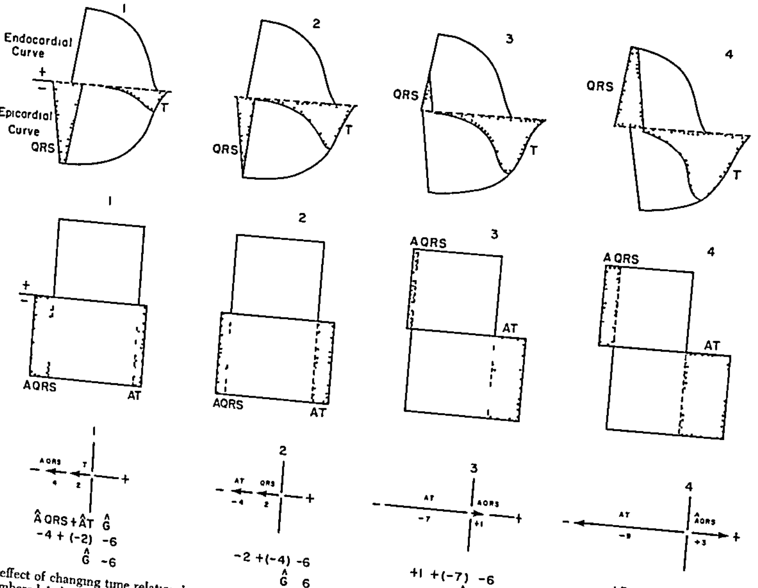


Fig 63 —The effect of changing time relationships of the epicardial and endocardial monophasic curves on the gradient In this figure each pair of monophasic curves numbered 1 through 4 is represented by a corresponding pair of rectangles to simplify calculation of the gradient G Thus the monophasic curves in column 1 if depicted as rectangles having the same area relatively as the monophasic curves themselves yield Â QRS and Â T which can be assigned the unit values of −4 and −2 Vector addition or algebraic addition of these values yields a gradient vector of −6 units As long as the areas enclosed by the two rectangles remain unchanged the time relationship between epicardial and endocardial curves or rectangles can be varied widely to produce marked changes in the direction and area of Â QRS and Â T (columns 2 3 and 4) However in every instance the gradient remains −6 units in magnitude and has the same direction as in column 1 Therefore the Â T changes in this figure are all secondary Â T changes consequent to alterations in cellular depolarization

polarization countless elementary QRS and T forces of differing magnitude and direction are produced by all the minute wedges of muscle forming the ventricular wall For each pair of QRS and T forces or vectors so produced there exists a corresponding gradient vector which expresses the net electrical effect of the different rate of recovery throughout the wedge of muscle

However in terms of body surface potentials as previously explained the elementary QRS and T
t —

By the same token for each succeeding pair of mean instantaneous QRS and T vectors there is a mean instantaneous ventricular gradient vector which represents the net electrical effects of the different rates of recovery in all muscle units of the ventricular wall simultaneously undergoing first depolarization and then repolarization Since the processes of depolarization and repolarization do not occur *simultaneously* in all portions of the ventricular muscle but *involve* the different regions sequentially the mean instantaneous QRS T and ventricular gradient spatial vectors vary somewhat in direction and magnitude from instant to instant during the cardiac cycle At present there is no practicable way to determine the instant-to-instant change in the mean instantaneous ventricular —

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spati... (sÂ QRS) and the mean T spatial vector (*sÂ T*) the mean ventricular gradient spatial vector (sĜ) The spatial vector of the ventricular gradient has been studied primarily in its frontal plane projection (Ĝ) which is the vectorial sum of the vectors representing the mean manifest electrical axis of QRS (Â QRS) and the mean electrical axis of T (Â T) The method used to calculate the mean ventricular gradient (Ĝ) from the electrocardiogram (see Fig 64) is outlined below

1 The amplitude and duration of the QRS deflection and T wave are measured in at le...

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2 The product of the amplitude of a deflection multiplied by one half its duration or width equals the area of the deflection in microvolt seconds This value is then converted into Ashman units (1 Ashman unit = 4 microvolt seconds) If either the QRS or the T deflection has two or more wave components the areas of these waves are measured separately and then added algebraically according to their sign to obtain the resultant area of the deflection

3 The values for the areas of the QRS and T deflections in the two leads are plotted on the appropriate lead axes of the triaxial reference frame and perpendiculars are dropped from the plotted points

4 Lines drawn from the center of the reference figure to the point of intersection of each pair of perpendicular lines represent the *mean manifest electrical axes* of QRS and T or Â QRS and Â T

5 The parallelogram method of vector addition is utilized to obtain the gradient vector which is the resultant of Â QRS and Â T Thus Â QRS and Â T serve as two sides of the parallelogram and the other two sides are formed by lines drawn parallel to the former A diagonal line drawn from the center of the reference figure represents the mean ventricular gradient (Ĝ)

Normally the mean ventricular gradient vector Ĝ lies within 30 and to the left or right of Â QRS in the frontal plane depending on whether the heart is in horizontal or vertical heart position In neither case does Ĝ normally deviate from Â QRS by an angle greater th n 30 T...

...st be added to that of the T wave if S-T segment deviations are present since the period during which the ventricles repolarize corresponds to both the S-T and the T intervals It is evident however that S-T segment deviations due to the current of injury produce significant error when introduced into the calculation of the ventricular gradient

The practical as well as theoretical importance of the ventricular gradient concept is that it provides a means of differentiating primary and secondary T wave variations These two types of T wave changes were explained earlier in terms of the hypothetical heart muscle cell but the basis for distinguishing one from the other is the same in clinical electrocardiography Thus the important relationship of T ...

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... mean axis of T can be altered by a change either in the mean ventricular gradient vector Ĝ or in the mean electrical axis of QRS Primary T wave changes result solely from a

in the duration of the excited state at these two surfaces, the gradient remains the same despite variations in the time sequence of onset of depolarization throughout the cell

The concepts demonstrated in this hypothetical experiment are of great importance in clinical electrocardiography and can be stated in a simplified way as follows (1) T wave (repolarization) changes which are not accompanied by a change in the ventricular gradient are due to some alteration in the time sequence of onset of depolarization throughout the cell or, more generally, throughout heart muscle

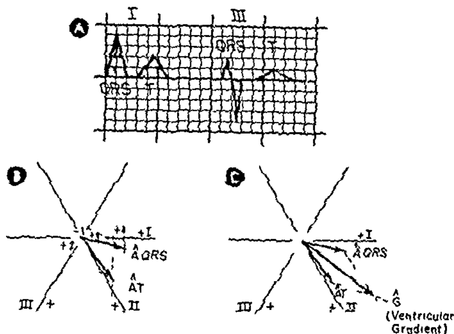


Fig 64 —Construction of the mean ventricular gradient vector Ĝ from the standard limb leads of the electrocardiogram The area of each QRS and T deflection in at least two of the three bipolar limb leads is calculated (A) utilizing the formula Area = ½ Width × Height and is expressed either in microvolt seconds or in Ashman units (1 Ashman unit = 4 microvolt seconds) In B, the values for the QRS deflections in leads I and III are plotted on appropriate axes of the triaxial reference frame and perpendiculars are dropped from the two points The vector drawn from the center of the reference figure to the point of intersection of the perpendicular lines represents Â QRS The same procedure is followed in determining Â T In C Â QRS and Â T are added vectorially by the parallelogram method to yield the resultant vector Ĝ the mean ventricular gradient vector

They are designated *secondary* T wave changes (2) T wave (repolarization) changes which are accompanied by an alteration in the direction and magnitude of the gradient vector are *primary* T wave abnormalities and reflect a change in the duration of the excited state in some region of the cell or of the heart muscle

Up to this point in the discussion the gradient concept has been considered only in its application to the rudimentary electrical activities of a single muscle cell However as will be recalled the hypothetical muscle cell is essentially the equivalent of a minute wedge of muscle extending through the entire thickness of ventricular wall since only the electrical effects of depolarization and repolarization at the epicardial and endocardial surfaces of heart muscle need be considered During ventricular depolarization and repolarization for each tiny wedge of ventricular muscle involved there are produced QRS and T forces which represent the algebraic sum of paired endocardial and epicardial monophasic curves specific for the muscle segment in question However, in the heart muscle cell in situ the duration of the excited state or the rate of recovery is not uniform through the cell as was the case in the hypothetical cell considered previously, nor is the duration of the excited state the same throughout the ventricular muscle as a whole In both instances the rate of recovery is more rapid in regions oriented toward the epicardium and slower in regions oriented toward the endocardium The reason for the more rapid rate of recovery in the subepicardial ventricular muscle of the normal human heart is not clear but it has been suggested that the greater pressure applied to inner layers of ventricular muscle during mechanical cardiac systole causes a delay in subendocardial recovery This permits the subepicardial muscle to recover first even though it is activated later than the deeper layers of ventricular muscle In any case it is evident that during ventricular depolarization and re

case when the hypothetical cell was stimulated at its epicardial end so also in bundle branch block the change in the depolarization process must inevitably produce a secondary change in the repolarization

tions producing primary T wave changes Â QRS and Â T show an equal but oppositely directed increment in size Thus the ventricular gradient vector **C** does not change in direction and if normal before onset of the bundle branch block it remains so If it should

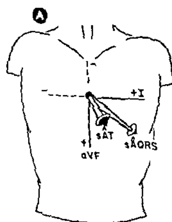
In this event, the presence of primary T wave abnormalities in addition to the secondary T wave changes of bundle branch block can be assumed This fact in particular potentially provides one of the more valuable applications of the ventricular gradient concept

Unfortunately there are at present limitations inherent to the methods used to determine the ventricular gradient which greatly impair its clinical usefulness The accuracy and reliability of frontal plane ventricular gradient calculations can be assured to some extent only by the most tedious and time-consuming measurements of specially recorded electrocardiograms The manual construction of the horizontal mean ventricular gradient vector and the mean ventricular gradient spatial vector introduces even greater inaccuracies However Briller and his associates have constructed an electronic analogue computer which automatically calculates from the vector cardiogram the magnitude and direction of the spatial ventricular gradient in terms of its projection on each of the three recording planes of the body

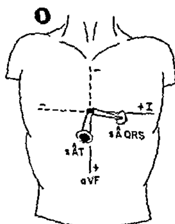
The Monophasic Curves and Ventricular Gradient in Myocardial Ischemia

As already indicated one can imagine that there exists a pair of monophasic curves for each minute wedge of ventricular muscle Thus for a given area of the ventricle or for the ventricular muscle mass as a whole there are endocardial and epicardial monophasic curves which represent the algebraic summation of all the monophasic curves of the component muscle units Since normally the rate of recovery is more rapid in epicardial muscle even though it is activated last the epicardial monophasic curve is therefore of shorter duration than the endocardial curve and is preceded by it Accordingly the QRS deflection and T wave recorded in a lead overlying the electrically dominant left ventricle tend to have the same upright direction Since subendocardial ischemia prolongs the endocardial monophasic curve so

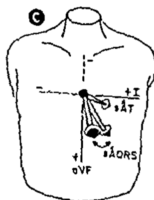
Fig 66 sÂ QRS ar emphified lel ob yo tec sÂ



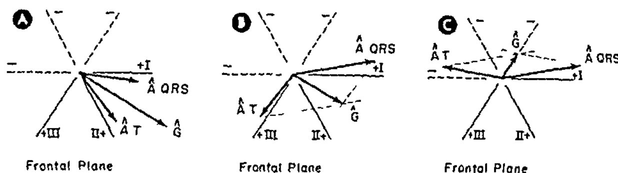
Normal Adult



Normal Elderly Adult



Normal Child



corded in lead I they would constitute secondary T
and the orientation of the ventricular gradient vector
has complicated the previously existent left bundle br
was the case in B however in this instance the ventricular gradient would be abnormal and the T wave abnormalities would be categorized as primary T wave changes (due to myocardial ischemia for example)

change in the direction or magnitude of the mean ventricular gradient vector i e from some change in the *rate of recovery and duration of the excited state in* various portions of the ventricular muscle Consequently a primary T wave change which is identified by the abnormal direction and/or magnitude of Ĝ is usually indicative of some myocardial abnormality A secondary change in the size or direction of the T wave is consequent to an alteration in the process of ventricular depolarization and is evidenced by a *change in the mean electrical axis of QRS and by a* normal ventricular gradient A secondary T wave change is not in itself indicative of myocardial abnormality although the basic disturbance of depolarization responsible for the T wave change may well imply this

Primary T wave changes include T wave abnormalities noted in the following myocardial ischemia electrolyte imbalance ventricular hypertrophy according to some authors quinidine and digitalis therapy and miscellaneous conditions like hypothyroidism *beriberi and other diseases not primarily involving* the heart

The lowering of the T wave amplitude or T wave inversion which is produced in normal subjects by digitalis and occasionally by exercise is somewhat unique in that it probably results from an increase in the rate of recovery of ventricular muscle the effect being more marked in regions with a slower rate of recovery normally Thus as the rate of recovery in subendocardial layers of ventricular muscle increases and approaches that of subepicardial myocardium the normal *gradient in* the duration of the excited state across the ventricular wall diminishes This is accompanied by a progressive decrease in the magnitude of the mean ventricular gradient vector (without any change in its direction) which in turn causes *progressive lowering and finally inversion of the T* waves in leads previously registering upright T waves For the same reason leads previously recording inverted T waves show upright T waves These T wave changes must of necessity occur, because Â T = Ĝ − Â QRS In this equation Â QRS does not change but Ĝ becomes smaller so that Â T must also become smaller until when finally Â QRS exceeds Ĝ the di*rection of Â T is reversed By and large changes in Â* T which are due to changes in magnitude of the ventricular gradient with essentially no change in its direction have much less significance in terms of cardiac disease than do directional changes in the gradient when these are not the result of changes in cardiac position

Secondary T wave changes are perhaps best illustrated by the T wave abnormalities characterizing bundle branch block From a theoretical standpoint bundle branch block is roughly analogous to the *hypothetical cell with a normal gradient in* duration of the excited state which is stimulated at its epicardial surface rather than at its endocardial surface This changes the time sequence of onset of depolarization throughout the cell The same end result is effected in bundle branch block but in this condition the causative mechanism is blocked impulse conduction down one of the main bundle branches Excitation can only reach the ventricular muscle on the blocked side by spreading from the intact bundle branch via the muscle fibers Consequently the time sequence of onset of depolarization in the blocked ventricle differs from that present during normal intraventricular conduction Just as was found to be the

case when the hypothetical cell was stimulated at its epicardial end so also in bundle branch block the change in the depolarization process must inevitably produce a *secondary* change in the repolarization process and therefore a change in the appearance of the T wave However if the bundle branch block is not complicated by cardiac disease or by other conditions producing primary T wave changes Â QRS and Â T show an equal but oppositely directed increment in size Thus the ventricular gradient vector G does not change in direction and if normal before onset of the b ndle branch bl l

63) *In this event* the presence of primary T wave abnormalities in addition to the secondary T wave changes of bundle branch block *can be assumed* This fact in particular potentially provides one of the more valuable applications of the ventricular gradient concept.

Unfortunately there are at present limitations inherent to the methods used to determine the ventricular gradient which greatly *impair its clinical usefulness* The accuracy and reliability of frontal plane ventricular gradient calculations can be assured to some extent, only by the most tedious and time-consuming measurements of specially recorded electrocardiograms The manual construction of the horizontal mean ventricular gradient vector and the mean ventricular gradient spatial vector introduces even greater inaccuracies However Briller and his associates have constructed an electronic analogue computer which automatically calculates from the vectorcardiogram the magnitude and direction of the spatial ventricular gradient in terms of its projection on each of the three recording planes of the body

The Monophasic Curves and Ventricular Gradient in Myocardial Ischemia

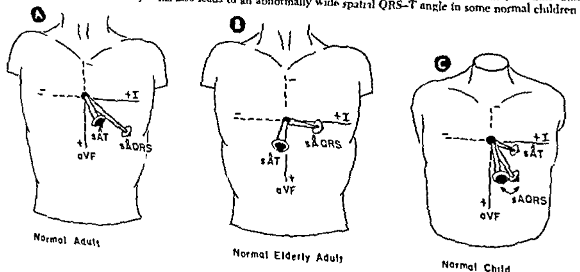
As already indicated one can imagine that there exists a pair of monophasic curves for each minute wedge of ventricular muscle Thus for a given area of the ventricle or for the ventricular muscle mass as a whole there are endocardial and epicardial monophasic curves which represent the algebraic summation of all the monophasic curves of the component muscle units Since normally the rate of recovery is more rapid in epicardial muscle even though it is activated last the epicardial monophasic curve is therefore of shorter duration than the endocardial curve and is preceded by it Accordingly the QRS deflection and T wave recorded in a lead overlying the electrically dominant left ventricle tend to have the same upright direction Since subendocardial ischemia prolongs the endocardial monophasic curve so

Fig 66 —The spatial QRS–T a

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ie anteriorly than in the adult and

ns also leads to an abnormally wide spatial QRS–T angle in some normal children



that it extends even farther behind the epicardial curve algebraic addition of the two curves yields a T wave of greater size but of unchanged direction As will be seen later in Chapter 18 this is indeed what occurs in subendocardial ischemia that is an overly[illegible] T [illegible] wh[illegible] changes in direction On the other hand when ischemia becomes transmural and extends to the epicardium the epicardial monophasic curve is prolonged beyond the endocardial curve so that algebraic addition of the two yields a T wave which is directed just the opposite of the QRS deflection that is to say an overlying lead will register an upright QRS deflection and an inverted T wave The greater the degree of slowing of recovery at the epicardial surface the larger and the more deeply inverted is the T wave in this lead

As will be indicated in Chapter 18 the S–T segment deviation which signals the appearance of muscle injury has much the same mechanism of production as the monophasic curves recorded from the single cell in the hypothetical experiment at the beginning of this chapter It will be recalled that a monophasic curve was recorded when depolarization was blocked at one or the other surface of the cell by cooling In the case of muscle injury the injury process itself has the same blocking effect on depolarization so that at the end of the QRS interval the injured region retains its dipoles and assumes a positive charge with respect to uninjured depolarized muscle elsewhere Consequently an overlying lead records an upward displacement of the base line during the S–T interval When at times the R wave S–T segment and T wave fuse to form a single wave the latter is often referred to as a *monophasic curve* Ischemia and injury will be discussed in greater detail later in Chapter 18 but there the electrical effects of these processes will be explained in vector terms rather than in terms of monophasic curves However the two approaches to the subject of ischemia and injury are fundamentally equivalent

The Spatial QRS–T Angle

From previous considerations of the ventricular repolarization process in the human heart and the ventricular gradient concept it readily follows that mean instantaneous T spatial vectors normally are oriented more or less in the same direction as the mean instantaneous QRS spatial vectors Accordingly the resultants of these instantaneous vectors the mean T and mean QRS spatial vectors (sÂ T and sÂ QRS) normally subtend a relatively narrow angle Grant and Estes have applied this fact to the clinical interpretation of electrocardiograms by utilizing their "cylinder method" of constructing mean spatial vectors from the electrocardiographic lead deflections to calculate the *spatial QRS–T angle* They found that in normal adult subjects the calculated spatial QRS–T angle is usually less than 40° and rarely exceeds 50° (Fig 66) The exceptions to this rule are normal elderly subjects whose spatial QRS–T angles will occasionally exceed 60° in the absence of detectable heart disease The spatial QRS–T angle in children and in many adolescents normally exceeds 60° Grant and Estes maintain that if the spatial QRS–T angle is used in interpreting electrocardiograms there is little to be gained from calculating the mean (frontal plane) ventricular gradient but this is open to question As these investigators themselves admit the spatial QRS–T angle does not distinguish between primary and secondary T wave abnormalities which is one of the prime functions of the ventricular gradient On the other hand the practical usefulness of the ventricular gradient is at present limited by the cumbersome calculations required for its determination although this handicap will undoubtedly be overcome in the future by the development of methods of electronically computing the ventricular gradient

For the present determination of the spatial QRS–T angle provides a practicable means of applying certain principles of the ventricular gradient concept to the clinical interpretation of the electrocardiogram However one should not conceive of the spatial QRS–T angle as representing the complete realization of the potentialities of the ventricular gradient concept although it does teach one to evaluate for example the T waves in relationship to the QRS deflections appearing in the same lead By computing the spatial QRS–T angle the electrocardiographer will find it much easier to differentiate normal T waves from abnormal T waves Granting these advantages of the spatial QRS–T angle one must at the same time call attention to certain disadvantages of this approach

1 Sometimes the null contour or transitional pathway for the mean T spatial vector does not pass through the electrode positions of any of the six precordial leads routinely recorded In this instance upright T waves are registered in all six chest leads For this to happen the horizontal mean T (planar) vector must lie in the +30 to +90 segment of the horizontal reference frame (this is as precise a localiza

tion of the vector as one can make with any degree of certainty.) Obviously the spatial QRS–T angle would have relatively little reliability in this situation unless one were to record additional precordial leads so that the null contour for the mean T spatial vector might be mapped out.

2. When the method of Grant and Estes is used to determine the spatial QRS–T angle there must be available a vector model of the type used by these investigators.

3. If, on the other hand, the orientations of sÂ QRS and sÂ T in the frontal and horizontal planes are determined separately for each plane, these values must then be used to calculate trigonometrically the spatial QRS–T angle by means of tables of the type devised by Helm (see Table 2).

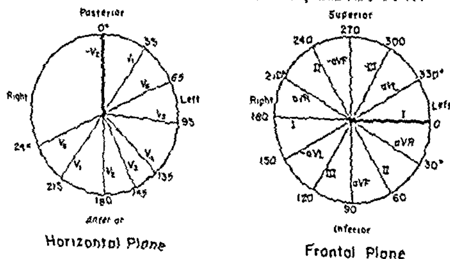
In either case determination of the spatial QRS–T angle is time consuming.

4. In determining the spatial QRS–T angle the effective direction and length of each individual lead is not taken into consideration. The implications of this fact have already been considered in Chapter 4 dealing with the lead vector concept. Suffice to say the use of reference systems based on the anatomic lead axes rather than on the corresponding lead vectors introduces some error into the construction of mean vectors from the electrocardiogram. Admittedly this factor may have relatively little significance except in cases of borderline wide spatial QRS–T angles.

5. Not all investigators will agree as to the correctness of the normal limits of the spatial QRS–T angle proposed by Grant and Estes; in fact some have found rather marked variation in the width of the spatial QRS–T angle in normal subjects.

Despite the objections just listed the spatial QRS–T angle represents a major step away from the empiricism with which the electrocardiographic T waves were interpreted in previous years.

TABLE 2.—CALCULATION OF THE SPATIAL ANGLE SUBTENDED BY sÂ QRS AND sÂ T OR BY THE LONG AXES (MAXIMAL INSTANTANEOUS SPATIAL VECTORS) OF THE QRS sE AND T sE LOOPS



	0	5	10	15	20	25	30	35	40	45	50	55	60	65	[illegible]				
	180	175	170	165	160	155	150	145											
	180	185	190	195	200	205	210	215											
	360	355	350	345	340	335	330	325											
cos 1.000																			
0°, 180, 180, 360; cos ≈ .9994	1.00	1.00	98	97	94	91	8	8	77	[illegible]	64	57	50	4	51	0	17	00	00
	00	09	17	0	64	4	50	57	64	71	77	8	8	91	91	97	95	1.00	1.00
	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	01	00
5°, 175, 355, 355	1.00	1.00	98	97	93	91	87	82	4	71	64	57	50	4	31	0	17	00	00
	00	09	1	26	42	4	50	57	64	1	0	8	86	90	93	96	95	00	1.00
	00	01	0	6	3	04	04	03	06	06	0	0	08	03	04	03	04	02	00

(*Continued on next page*)

that it extends even farther behind the epicardial curve, algebraic addition of the two curves yields a T wave of greater size but of unchanged direction. As will be seen later in Chapter 18, this is indeed what occurs in subendocardial ischemia; that is, an overlying lead registers a taller T wave. This is a primary T wave change, since Â QRS does not change in area while Â T does—hence the ventricular gradient also changes in direction. On the other hand, when ischemia becomes transmural and extends to the epicardium, the epicardial monophasic curve is prolonged beyond the endocardial curve so that algebraic addition of the two yields a T wave which is directed just the opposite of the QRS deflection, that is to say, an overlying lead will register an upright QRS deflection and an inverted T wave. The greater the degree of slowing of recovery at the epicardial surface, the larger and the more deeply inverted is the T wave in this lead.

As will be indicated in Chapter 18, the S–T segment deviation which signals the appearance of muscle injury has much the same mechanism of production as the monophasic curves recorded from the single cell in the hypothetical experiment at the beginning of this chapter. It will be recalled that a monophasic curve was recorded when depolarization was blocked at one or the other surface of the cell by cooling. In the case of muscle injury, the injury process itself has the same blocking effect on depolarization, so that at the end of the QRS interval the injured region retains its dipoles and assumes a positive charge with respect to uninjured depolarized muscle elsewhere. Consequently, an overlying lead records an upward displacement of the base line during the S–T interval. When at times the R wave, S–T segment and T wave fuse to form a single wave, the latter is often referred to as a *monophasic curve*. Ischemia and injury will be discussed in greater detail later in Chapter 18, but there the electrical effects of these processes will be explained in vector terms rather than in terms of monophasic curves. However, the two approaches to the subject of ischemia and injury are fundamentally equivalent.

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From previous considerations of the ventricular repolarization process in the human heart and the ventricular gradient concept, it readily follows that mean instantaneous T spatial vectors normally are *oriented more or less in the same direction* as the mean instantaneous QRS spatial vectors. Accordingly, the resultants of these instantaneous vectors, the mean T and mean QRS spatial vectors (sÂ T and sÂ QRS), normally subtend a relatively narrow angle. Grant and Estes have applied this fact to the clinical interpretation of electrocardiograms by utilizing their "cylinder method" of constructing mean spatial vectors from the electrocardiographic lead deflections to calculate the *spatial QRS–T angle*. They found that in normal adult subjects the calculated spatial QRS–T angle is usually less than 40° and rarely exceeds 50° (Fig. 66). The exceptions to this rule are normal elderly subjects whose spatial QRS–T angles will occasionally exceed 60° in the absence of detectable heart disease. The spatial QRS–T angle in children and in many adolescents normally exceeds 60°. Grant and Estes maintain that if the spatial QRS–T angle is used in interpreting electrocardiograms, there is little to be gained from calculating the mean (frontal plane) ventricular gradient, but this is open to question. As these investigators themselves admit, the spatial QRS–T angle does not distinguish between primary and secondary T wave abnormalities, which is one of the prime functions of the ventricular gradient. On the other hand, the practical usefulness of the ventricular gradient is at present limited by the cumbersome calculations required for its determination, although this handicap will undoubtedly be overcome in the future by the development of methods of electronically computing the ventricular gradient.

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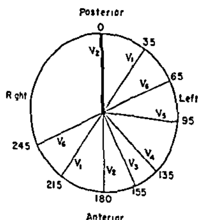
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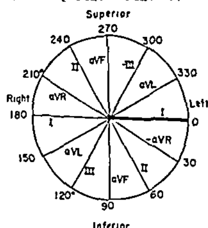
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TABLE 2 — CALCULATION OF THE SPATIAL ANGLE SUBTENDED BY sÂ QRS AND sÂ T OR BY THE LONG AXES (MAXIMAL INSTANTANEOUS SPATIAL VECTORS) OF THE QRS sÊ AND T sÊ LOOPS





	0	5	10	15	0	25	30	35	40	45	50	55	60	65	0	75	80	85	90
	(+)	(+)	(+)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(+)
	180	175	1 0	165	160	155	150	145	140	135	130	125	120	115	110	105	100	95	90
	(	(−+	(−)	(−+)	(−+)	(−+)	(−+)	(−)	(−+)	(−+)	(−+)	(−+)	(−+)	(−+)	(−+)	(−+)	(−+)	(−+)	(−+)
	180	185	190	195	200	05	210	215	220	225	230	235	240	245	250	255	260	263	270
	(−−)	(−)	(−−	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)	(−−)
	360	355	350	345	340	335	330	325	3?0	315	310°	305	300	95	290	85	250	275	2 0
	(−)	(−	(+−	−)	(+−)	(+−)	(+−)	(+−)	(+−)	(+−)	(+)	(+−)	(+−)	(+−)	(+−)	(+−)	(+−)	(+−)	(+−)
1 000																			
0 (+) 180°−	99	1 00	98	97	94	91	87	82	77	71	64	57	50	42	34	26	17	09	00
180°(−−)	00	09	17	6	34	42	50	57	64	71	77	82	87	91	94	97	98	1 00	1 00
360 (− n = 9994	00	00	00	00	09	00	00	00	00	00	00	00	00	00	00	00	00	09	00
5° (+) 175 ( )	1 00	1 00	98	97	94	91	87	82	6	71	64	57	50	42	34	26	17	09	00
185 (−−)	00	09	17	26	34	42	50	57	64	71	6	82	86	90	94	96	98	99	1 00
355 ( )	00	01	02	0	03	04	04	05	06	06	07	07	08	08	09	09	09	09	09

(*Continued on next page*)

TABLE 2 (*Continued*)

	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90
	(−−) 360 (+−)	(−−) 355 (+−)	(−−) 350 (+−)	(−−) 345 (+−)	(−−) 340 (+−)	(−−) 335 (+−)	(−−) 330 (+−)	(−−) 325 (+−)	(−−) 320 (+−)	(−−) 315 (+−)	(−−) 310 (+−)	(−−) 305 (+−)	(−−) 300 (+−)	(−−) 295 (+−)	(−−) 290 (+−)	(−−) 285 (+−)	(−−) 280 (+−)	(−−) 275 (+−)	(−−) 270 (+−)
cos = 991																			
10 (++)																			
170 (−+)	1 00	1 00	98	96	94	90	86	81	76	70	64	57	49	42	34	26	17	09	00
190 (−−)	00	09	17	26	34	42	50	57	64	70	76	81	86	89	93	95	97	98	98
350 (+−)	00	02	03	05	06	07	09	10	11	12	13	14	15	16	16	17	17	17	17
cos = 976																			
15 (++)																			
165 (−+)	1 00	1 00	98	96	94	90	86	81	75	69	63	56	49	41	33	25	17	08	00
195 (−−)	00	09	17	26	34	42	50	57	61	69	75	80	84	88	91	94	95	96	97
345 (+−)	00	02	05	07	09	11	13	15	17	19	20	21	23	24	24	25	26	26	26
cos = 954																			
0 (++)																			
160 (−+)	1 00	1 00	98	96	93	90	85	80	75	68	62	55	48	40	32	24	16	08	00
200 (−−)	00	09	17	26	34	42	49	56	63	68	74	79	83	86	89	91	93	94	94
340 (+−)	00	03	06	09	12	15	18	20	23	25	27	29	30	31	32	33	34	34	34
cos 974																			
25 (++)																			
155 (−+)	1 00	1 00	98	96	93	89	84	9	73	67	61	54	46	39	31	24	16	08	00
205 (−−)	00	09	17	26	34	41	49	55	62	67	72	77	80	83	86	88	89	90	91
335 (+−)	00	04	08	12	16	19	23	26	29	31	34	36	37	39	40	41	42	42	42
cos = 887																			
30 (++)																			
150 (−+)	1 00	99	98	96	92	88	83	78	72	65	57	52	45	37	30	23	15	08	00
210 (−−)	00	09	17	26	34	41	48	54	60	65	70	74	77	80	83	84	86	86	87
330 (+−)	00	05	10	15	19	24	28	31	35	38	40	43	45	46	48	49	49	50	50
cos = 813																			
35 (++)																			
145 (−+)	1 00	99	98	95	91	87	82	76	70	63	57	50	43	36	29	21	14	07	00
215 (−−)	00	09	17	25	33	41	47	53	59	63	68	71	74	77	79	80	81	82	82
325 (+−)	00	06	12	18	23	28	33	37	41	44	47	50	52	54	55	56	57	57	57
cos = 793																			
40 (++)																			
140 (−+)	1 00	99	97	94	90	85	80	74	67	61	54	47	40	34	27	20	13	07	00
220 (−−)	00	09	17	25	33	40	47	52	57	61	64	68	70	72	74	75	76	76	77
320 (+−)	00	07	14	21	28	33	39	43	47	51	54	57	59	61	62	63	64	64	64
cos = 737																			
45 (++)																			
135 (−+)	1 00	99	97	94	89	81	77	71	64	58	51	44	38	31	25	19	12	06	00
225 (−−)	00	09	17	25	32	39	45	50	54	58	61	63	65	67	68	69	0	71	71
315 (+−)	00	09	17	25	32	39	45	50	54	58	61	63	65	67	68	69	70	71	71
cos = 676																			
50 (++)																			
130 (−+)	1 00	99	96	92	87	81	74	68	61	54	47	41	35	29	23	17	11	06	00
230 (−−)	00	09	17	25	32	38	43	47	51	54	57	59	60	62	63	63	64	64	64
310 (+−)	00	10	20	29	38	45	51	56	61	64	67	70	72	73	75	75	76	76	77
cos = 609																			
55 (++)																			
125 (−+)	1 00	99	96	91	84	78	70	63	56	50	43	37	31	26	20	15	10	05	00
235 (−−)	00	09	17	24	31	36	41	44	47	50	53	53	54	55	56	57	57	57	57
305 (+−)	00	12	24	35	44	52	58	63	68	71	74	76	78	79	80	81	81	82	82
cos = 537																			
60 (++)																			
120 (−+)	1 00	99	94	88	81	73	65	58	51	43	39	33	28	23	18	13	09	04	00
240 (−−)	00	09	17	24	29	34	38	41	43	43	46	47	48	49	49	50	50	50	50
300 (+−)	00	15	29	41	51	59	65	0	74	77	80	82	83	84	85	86	86	87	87
co = 462																			
65 (++)																			
115 (−+)	1 00	98	92	84	76	67	59	52	45	39	33	28	24	19	15	11	07	04	00
245 (−−)	00	09	16	23	28	31	34	36	38	39	40	41	41	41	42	42	42	42	42
295 (+−)	00	18	35	49	59	67	73	78	81	83	85	87	88	89	90	90	90	91	91
cos = 383																			
70 (++																			
110 (−+)	1 00	97	89	79	68	59	51	44	38	32	28	23	19	16	12	09	06	03	00
250 (−−)	00	08	16	20	25	28	29	31	32	32	33	33	34	34	34	34	34	34	34
290 (+−)	00	23	43	58	68	76	81	84	87	89	90	91	92	93	93	94	94	94	94
co = 301																			
75 (++)																			
105 (−+)	1 00	95	83	69	58	49	41	35	29	25	21	18	15	12	09	07	05	02	00
255 (−−)	00	08	15	19	20	23	24	24	25	25	25	25	26	26	26	26	26	26	26
285 (+−)	00	31	54	69	79	84	88	91	92	94	94	95	96	96	96	96	96	97	97
s = 216																			
80 (++)																			
100 (−+)	1 00	89	0	54	43	35	29	24	20	17	14	12	10	08	06	05	03	0	00
260 (−−)	00	08	12	15	16	16	17	17	17	17	17	17	17	17	17	17	17	17	17
280 (+−)	00	44	70	83	89	92	94	96	96	97	97	98	98	98	98	98	98	98	98
cos = 133																			
85 (++)																			
95 (−+)	1 00	71	44	31	23	18	15	12	10	09	07	06	05	04	03	02	02	01	00
265 (−−)	00	06	08	08	08	09	09	09	09	09	09	09	09	09	09	09	09	09	09
275 (+−)	00	71	83	95	97	98	99	99	99	99	99	99	99	1 00	1 00	1 00	1 00	1 00	1 00
co = 044																			
90 (++)																			
90 (−+)	—	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00
270 (−−)	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00
270 (+−	—	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00	1 00
co = 000																			

TABLE 2 (*Continued*)

The orientations of sÂ QRS and sÂ T as expressed in terms of the reference frames used throughout this text

tial angle between the QRS and T vectors the orientation of the QRS vector in the horizontal plane is located in the horizontal marginal columns at the top of the table while the orientation of the QRS vector in the frontal plane is located in the vertical marginal column at the left of the table The three numbers appearing at the intersection of the columns are read directly from the table and positive

respectively The two signs obtained from the vertical marginal columns are inserted before the middle and lower numbers respectively The sign of the middle number should be the same in both instances or some error in the orientations of the vectors has been made The same procedure is followed with reference to the T vector Thus one is left with two sets of three values The paired upper values the paired middle values and the paired lower values are multiplied and the three products are then added algebraically according to their signs The result is the cosine of the angle between the two spatial vectors In the table cosines for values midway between the groups of angles given in the vertical marginal columns are listed By locating in these columns the position of the calculated cosine the spatial angle can be determined to the nearest 5 If the calculated cosine is positive the angle chosen must be between 0 and 90 If the calculated cosine has a negative value the angle chosen must be between 90 and 180 since the angle between two spatial vectors cannot exceed 180

Example (Fig 53 Chap 5)

	Horizontal Plane	Frontal Plane
Orientation* of sÂ QRS	65	60
Orientation of sÂ T	125	30

QRS T
+0 23 × 0.52 = −0 1196
+0 49 × +0 74 = +0 3626
+0 84 × +0 43 = +0.3612
+0 604_

Cosine of spatial angle subtended by sÂ QRS and sÂ T = +0 6042
Spatial QRS–T angle — approximately 55

Example (Fig 83 A, Chap 7)

	Horizontal Plane	Frontal Plane
Orientation of long axis of QRS sE loop	80	45
Orientation of long axis of T sE loop	100	40

QRS T
+0 12 × −0 13 = −0 0156
+0 70 × +0 76 = +0.5320
+0 70 × +0 64 = +0 4480
+0 9644

C

+0 9644

—

rms the ce f mes depicted in the box table

Vectorcardiography

INSTRUMENTATION

THE CATHODE RAY TUBE of the oscilloscope electrocardiograph and vectorcardiograph functions in the following way. An electrically heated filament enclosed in an evacuated glass tube heats a metal plate cathode covered with some substance capable of emitting electrons freely when hot. A second plate, the anode, is maintained at a high positive potential by an outside current source. As a result, a strong electrical field exists between the cathode and anode, and in this field the electrons emitted by the cathode are accelerated to a high velocity. The electrons pass through a small hole in the anode and emerge beyond as a fine beam of cathode rays. This electron beam passes between two sets of deflection plates at right angles to each other and strikes a fluorescent viewing screen on the inner surface of the oscilloscope tube. As long as the deflection plates remain uncharged, the electron beam remains focused at a single point on the screen, but once a potential difference appears between the two pairs of plates, the electron beam is drawn toward the positively charged plate of each pair and deflected away from the negatively charged plate. *Superior-inferior displacement of the beam is* governed by the potential difference across the vertical (Y) deflecting plates, and side-to-side displacement of the beam by the potential difference across the transverse or horizontal (X) plates.

*When the oscilloscope is used to record an electro*cardiogram, electrical currents from the patient are transmitted by lead wires to the oscilloscope, where, after considerable amplification, they produce a potential difference across the vertical deflection plates. *If the superior plate is relatively positively charged* with respect to the inferior plate, the electron beam is displaced upward; if the inferior plate is more positive or less negative than the superior plate, the beam moves downward. At the same time that the *potential variations across* the plates deflect the beam vertically upward and/or downward, a positive charge is placed intermittently on the left horizontal deflection plate, causing the electron beam to sweep at a given speed horizontally from right to left across the screen (i.e., from the observer's left to right). Thus the voltage recorded in a single electrocardiographic lead is depicted as a function of time.

When the oscilloscope is used as a vectorcardiograph, the horizontal beam sweep is turned off and two leads, preferably with perpendicular lead axes, are connected to the vertical and horizontal pairs of deflection plates. Thus, at a given instant during electrical systole or diastole, the electron beam is displaced according to the resultant of the cardiac forces acting along the two perpendicular leads being recorded and across the two pairs of deflection plates. If one visualizes the cardiac forces acting along the leads as vectors, then, in a general sense, the distance (translated into millivolts according to the standardization factors used for the two leads) the electron beam is displaced and the direction of displacement correspond roughly to the magnitude and direction of the *resultant or mean instantaneous* planar vector. The instantaneous planar vector itself can be visualized as extending from the point of origin, or resting point of the beam, to the position of the beam following displacement.

The instant-to-instant change in the direction and magnitude of the instantaneous cardiac vectors during atrial and ventricular depolarization and during ventricular repolarization is accompanied in the vectorcardiogram by the inscription of three successive closed loops—the P s$\hat{E}$,* QRS s$\hat{E}$, and T s$\hat{E}$ loops, respectively. To provide a means of timing in the vectorcardiogram, the electron beam is interrupted by an

*E = equivalent cardiac dipole. $\hat{E}$ = E expressed as vector quantity, and s$\hat{E}$ = E expressed as spatial vector quantity.

oscillator circuit which breaks the loops into dashes In the vectorcardiograms appearing in this text the dashes occur at intervals of 0 0025 second Each dash is modulated into the form of a teardrop the blunt end of which indicates the direction of inscription of a given loop The distance between the dashes indicates *the speed of inscription* Closely spaced dashes signify a slow rate of inscription widely spaced and elongated dashes a rapid speed of inscription

Since only two leads can be connected to the deflection plates of the oscilloscope at any single time the three loops traced on the oscilloscope screen during the cardiac cycle represent the projections of the *P sE, QRS sE* and *T sE* (spatial) loops on the plane defined by the axes of the leads being recorded In this text the loops visualized on the screen of the oscilloscope will be referred to either as the *planar loops* or more specifically as *frontal QRS loops* (sagittal P loop horizontal T loop etc) or the *QRS sF loop in the frontal projection* To obtain the frontal projection of the vectorcardiogram two leads which define the frontal plane are recorded preferably leads paralleling the transverse (X) and vertical (Y) *co-ordinate* axes of the body Similarly the *horizontal projection* can be obtained by recording a transverse lead and an anteroposterior lead (*Z*) and the sagittal projection can be obtained by recording vertical and anteroposterior leads

ELECTRODE PLACEMENT AND LEAD SYSTEMS

The lead systems used most commonly in vectorcardiography are of two types The first type includes all lead systems based on the Einthoven *equilateral* triangle such as those devised by Milovanovich Jouve Vastesaeger and the "equilateral tetrahedron" system suggested by Wilson and used most extensively by Burch and his associates The second general type of electrode arrangement *consists of the* orthogonal lead systems Of these the most widely used are Duchosal and Sulzer s rectangular system and Grishman s cube system a modification of the former Orthogonal lead systems differ from those based on the *equilateral* triangle in that the lead electrodes are applied in such a way that the leads formed are perpendicular to one another

Equilateral Tetrahedron Lead System

In the equilateral *tetrahedron* system electrodes are placed on the lef

[illegible]

the oscilloscope so as to record each of the planar projections of the vectorcardiogram (see Fig 68) is described below

Frontal plane—1 The lead wires from the left and right arms are connected to the horizontal deflection plates in such a way that when the right arm (RA) electrode is relatively *negative* with respect to the left arm (LA) electrode the electron beam is displaced to the subject s and screen s left (the observer s right)

2 The lead wire from the left leg (LL) is connected to the *inferior deflection* plate of the vertical set and Wilson s central terminal (*CT*) is connected to the superior deflecting plate When electrode LL is relatively positive with respect to CT the electron beam is deflected vertically downward

Left sagittal plane—1 Wilson s central terminal (*CT*) is connected to the horizontal deflection plate on the screen s right (the observer s left) and the lead wire from the back electrode (B) *is connected to the other horizontal* plate When B is relatively negative with respect to CT the electron *beam is* shifted anteriorly or to the screen s right (*the observer s left*)

2 The left leg electrode (LL) is *connected to the* inferior deflecting plate and CT to the superior plate

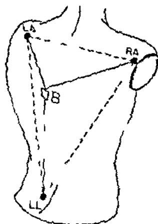


Fig 67 —Equilateral tetrahedron system of electrode placement used by *Burch* and his associates The v c g cardiogram is *recorded* in the *frontal* plane (defined by points LA RA and LL) *in* the left sagittal plane (defined by points *LA LL* and B the back *electrode*) and in the superior plane (defined by points LA RA and *B*)

Vectorcardiography

INSTRUMENTATION

THE CATHODE RAY TUBE of the oscilloscope electrocardiograph and vectorcardiograph functions in the following way. An electrically heated filament enclosed in an evacuated glass tube heats a metal plate cathode covered with some substance capable of emitting electrons freely when hot. A second plate, the anode, is maintained at a high positive potential by an outside current source. As a result, a strong electrical field exists between the cathode and anode, and in this field the electrons emitted by the cathode are accelerated to a high velocity. The electrons pass through a small hole in the anode and emerge beyond as a fine beam of cathode rays. This electron beam passes between two sets of deflection plates at right angles to each other and strikes a fluorescent viewing screen on the inner surface of the oscilloscope tube. As long as the deflection plates remain uncharged, the electron beam remains focused at a single point on the screen, but once a potential difference appears between the two pairs of plates, the electron beam is drawn toward the positively charged plate of each pair and deflected away from the negatively charged plate. Superior-inferior displacement of the beam is governed by the potential difference across the vertical (Y) deflecting plates, and side-to-side displacement of the beam, by the potential difference across the transverse or horizontal (X) plates.

When the oscilloscope is used to record an electrocardiogram, electrical currents from the patient are transmitted by lead wires to the oscilloscope, where after considerable amplification they produce a potential difference across the vertical deflection plates. If the superior plate is relatively positively charged with respect to the inferior plate, the electron beam is displaced upward; if the inferior plate is more positive or less negative than the superior plate, the beam moves downward. At the same time that the potential variations across the plates deflect the beam vertically upward and/or downward, a positive charge is placed intermittently on the left horizontal deflection plate, causing the electron beam to sweep at a given speed horizontally from right to left across the screen (i.e., from the observer's left to right). Thus the voltage recorded in a single electrocardiographic lead is depicted as a function of time.

When the oscilloscope is used as a vectorcardiograph, the horizontal beam sweep is turned off, and two leads, preferably with perpendicular lead axes, are connected to the vertical and horizontal pairs of deflection plates. Thus, at a given instant during electrical systole or diastole, the electron beam is displaced according to the resultant of the cardiac forces acting along the two perpendicular leads being recorded and across the two pairs of deflection plates. If one visualizes the cardiac forces acting along the leads as vectors, then in a general sense the distance (translated into millivolts according to the standardization factors used for the two leads) the electron beam is displaced and the direction of displacement correspond roughly to the magnitude and direction of the resultant or mean instantaneous planar vector. The instantaneous planar vector itself can be visualized as extending from the point of origin, or resting point of the beam, to the position of the beam following displacement.

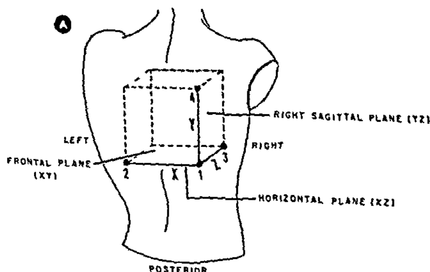
The instant-to-instant change in the direction and magnitude of the instantaneous cardiac vectors during atrial and ventricular depolarization and during ventricular repolarization is accompanied in the vectorcardiogram by the inscription of three successive closed loops—the P sĒ,* QRS sĒ, and T sĒ loops, respectively. To provide a means of timing in the vectorcardiogram, the electron beam is interrupted by an

*E = equivalent cardiac dipole. Ē = E expressed as vector quantity, and sĒ = E expressed as spatial vector quantity.

nomenclature used by Grishman and his associates to designate the leads will be changed so as to emphasize the relationship of the leads to the co-ordinate axes of the body Thus the three bipolar leads of the cube system will be assigned the symbols Xc* Yc and Zc Lead Xc is the transverse lead (Grishman's lead A) lead Yc is the vertical lead (Grishman's lead C) and lead Zc is the sagittal lead (Grishman's lead B) The corresponding body axes will be referred to as the X Y and Z axes just as in preceding chapters

The positions of the electrodes in the cube system and the polarity of the electrodes (Fig 69) follow

Lead Electrode	*Position*
Common electrode* (serves as the negative electrode for leads Xc and Zc and the positive electrode for lead Yc)	Right posterior axillary line at level of first or second lumbar vertebra (point 1 in Fig 69 A)
Positive electrode for lead Xc (transverse lead)	Left posterior axillary line at same level as the above (point 2 in Fig 69 A)
Positive electrode for lead Zc (sagittal or anteroposterior lead)	Right anterior axillary line at same level as the above (point 3 in Fig 69 A)
Negative electrode for lead Yc (vertical lead)	Over right scapula in the right posterior axillary line (point 4 in Fig 69 A)



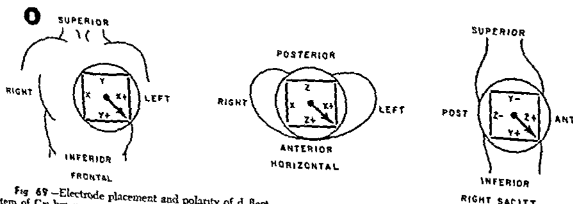


Fig 69 —Electrode placement and polarity of d Bae tem of Gr hm ... ube system of electrode placement

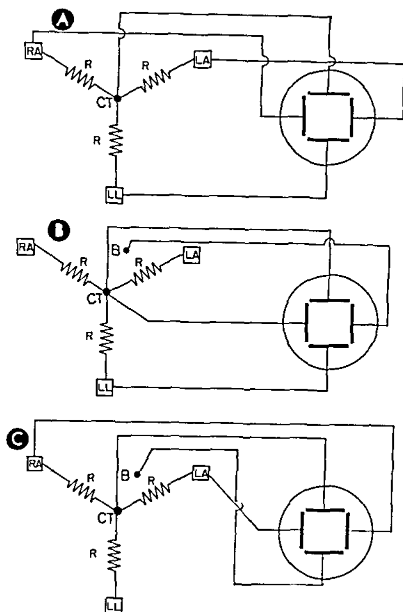


Fig 68 —Equilateral tetrahedron system of electrode placement The electrodes are connected to the deflection plates of the oscilloscope as follows **A** in recording the frontal projection of the vectorcardiogram **B** in recording the left sagittal plane projection and **C** in recording the superior projection *CT* central terminal of Wilson *B* a unipolar back lead placed at a point 2 cm to the left of the seventh dorsal vertebra Note that in recording the superior projection the connections of the left and right arms to the right and left deflection plates of the oscilloscope are the reverse of those utilized to record the frontal projection and that the normal QRS sÊ loop as recorded with this system tends to be situated to the observer s right in the frontal projection and to his left in the superior projection

the beam moving downward when LL is positive with respect to CT

Superior plane —The lead wire connections to the oscilloscope in recording the superior projection of the vectorcardiogram are shown in Figure 68 C and will not be described here

Standardizing factors —1 Horizontal deflection in frontal plane (R–L) is 1 unit (usually 1 inch) for 1 mv potential difference between electrodes R and L (See Chapter 2 for calculation of correction factors for unipolar leads when bipolar leads are assigned a value of 1)

2 Vertical deflection in frontal plane (C–F) is 1 7 units (or inches) per 1 mv potential difference between electrode *F* and Wilson s central terminal (See Chapter 2 for calculation of standardization factor for lead VF)

3 Horizontal deflection in sagittal plane (C–B) is 1 2 units (or inches) for 1 mv potential difference between the back electrode (*B*) and the Wilson s central terminal *

Grishman s Cube Lead System

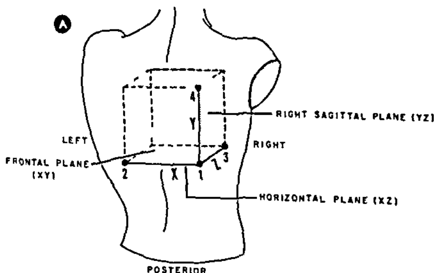
Grishman s *cube system of electrode placement* a modification of the rectangular system of Duchosal and Sulzer *is one of the orthogonal systems more* commonly used in this country It was the lead system utilized to record virtually all of the vectorcardiograms appearing in this text In the following description of the cube method of electrode placement the

*Abildskov J A Burch G E and Cronvich J A The validity of the equilateral tetrahedron as a spatial reference system Circulation 2 122 1950

nomenclature used by Grishman and his associates to designate the leads will be changed so as to emphasize the relationship of the leads to the co-ordinate axes of the body. Thus the three bipolar leads of the cube system will be assigned the symbols Xc, Yc and Zc. Lead Xc is the transverse lead (Grishman's lead A), lead Yc is the vertical lead (Grishman's lead C) and lead Zc is the sagittal lead (Grishman's lead B). The corresponding body axes will be referred to as the X, Y and Z axes just as in preceding chapters.

The positions of the electrodes in the cube system and the polarity of the electrodes (Fig 69) follow

Lead Electrode	*Position*
Common electrode* (serves as the negative electrode for leads Xc and Zc and the positive electrode for lead Yc)	Right posterior axillary line at level of first or second lumbar vertebra (point *1* in Fig 69 A)
Positive electrode for lead Xc (transverse lead)	Left posterior axillary line at same level as the above (point *2* in Fig 69 A)
Positive electrode for lead Zc (sagittal or anteroposterior lead)	Right anterior axillary line at same level as the above (point *3* in Fig 69 A)
Negative electrode for lead Yc (vertical lead)	Over right scapula in the right posterior axillary line (point *4* in Fig 69 A)



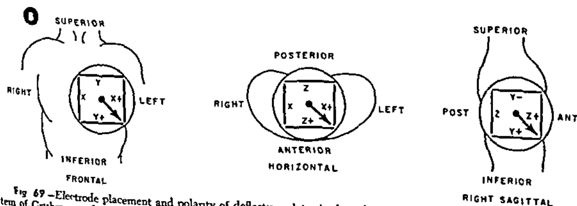


Fig 69 —Electrode placement and polarity of deflection plates in the cube orthogonal vectorcardiographic lead system of Grishman and his associates. A, positions of the electrodes. B, polarity of deflection plates of oscilloscope and standardization deflection.

Xc = transverse lead in the cube system of electrode placement

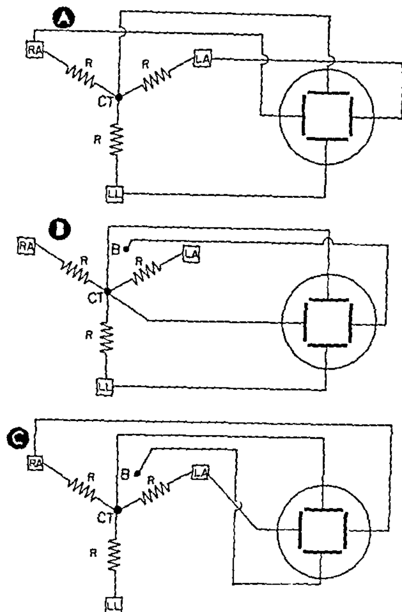


Fig 68 —Equilateral tetrahedron system of electrode placement The electrodes are connected to the deflection plates of the oscilloscope as follows **A** in *recording the frontal projection of the* vectorcardiogram **B** *in* recording the left sagittal plane projection and **C** in recording the superior projection *CT* central terminal of Wilson *B* a unipolar back lead placed at a point 2 cm to the left of the seventh dorsal vertebra Note that in recording the superior projection the connections of the left and right arms to the right and left deflection plates of the oscilloscope are the reverse of those utilized to record the frontal projection and that the normal QRS sE loop as recorded with this system tends to be situated to the observer s right in the frontal projection and to his left in the superior projection

the beam moving downward when LL is positive with respect to CT

Superior plane —The lead wire connections to the oscilloscope in recording the superior projection of the vectorcardiogram are shown in Figure 68 C and will *not be described here*

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2 *Vertical deflection in frontal plane (C–F) is* 1 7 units (or inches) per 1 mv potential difference between electrode F and Wilson s central terminal (See Chapter 2 for calculation of standardization factor for lead VF)

3 *Horizontal deflection in sagittal plane (C–B)* is 1 2 units (or inches) for 1 mv potential difference between the back electrode (B) and the Wilson s central terminal *

Grishman s Cube Lead System

Grishman s *cube system of electrode placement* a modification of the rectangular system of Duchosal and Sulzer is one of the orthogonal systems more commonly used in this country *It was the lead system* utilized to record virtually all of the vectorcardiograms appearing in this text In the following description of the cube method of electrode placement the

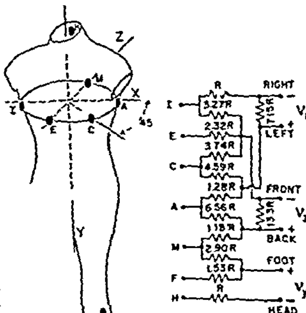
*Abildskov J A Burch G E and Cronvich J A The validity of the equilateral tetrahedron as a spatial reference system Circulation 2 122 1950

of the oscilloscope as can be seen in Figure 70

The theory underlying Grishman's cube lead system is as follows If the electrical center of the heart (*E*) is visualized as occupying the center of a sphere eight points on the surface of this sphere can be selected which are equidistant from the dipole center (*E*) and form the corners of a cube (Fig 71) In the cube method, three bipolar leads are applied to the thorax so as to form (in theory at least) three adjoining sides of a cube having *E* as its center The electrical center of the heart is assumed to occupy the center of a sagittal plane passing just to the left of the sternum in the fourth intercostal space Since the lengths of the leads their distances from the electrical center of the heart (*E*) and the angle each subtends with *E* were at one time all thought to be the equal, this system was originally considered by some authorities to be the most accurate method of recording the vectorcardiogram but this is certainly not the case

No attempt will be made in this text to evaluate the comparative merits of the equilateral tetrahedron and cube systems other than to point out that the equilateral tetrahedron is said to yield frontal plane vector loops which conform better with the findings in the limb leads of the scalar electrocardiogram On the other hand, the horizontal projection in the cube system correlates better with the precordial electrocardiogram Actually both the cube and tetrahedron lead systems are being superseded by lead systems which correctly record the magnitude and direction of the components of the spatial vector The theoretical basis of *corrected lead systems* such as the SVEC system of Schmitt and his associates and the vectorcardiographic system of Frank was outlined in limited detail in Chapter 4 The fact deserves emphasis that the corrected lead systems such as those just cited were devised, for the most part, from data obtained from torso model studies Nevertheless there is reason to believe that the information derived from these investigations—and for that matter the corrected lead systems as well—are valid when applied to the human torso and electrical potentials generated by the heart

Of the corrected lead systems which have been advocated we have favored the system proposed by Frank (Fig 72) since it entails the use of only seven electrodes as contrasted with the fourteen electrodes required by the SVEC system of Schmitt and his coworkers Moreover in our limited experience the Frank system has yielded quite satisfactory results In this system electrodes *A*, *C* *E* *I* and *M* are situated at the same transverse level (approximately the fifth intercostal space) Electrode *A* is placed in the left midaxillary line electrode *I* is placed in the right midaxillary line electrodes *E* and *M* are applied in the midlines anteriorly and posteriorly respectively and, finally electrode *C* is situated at an angle of 45 between anterior midline and left midaxillary line Electrode *F* is placed on the left leg and electrode *H* on



the back of the neck The X component of the spatial vector in the Frank lead system is derived from electrodes *A* *C* and *I* electrode *C* serving to introduce a correction for the backward slant of the image line or lead vector *I–A*. The Z, or anteroposterior component of the spatial vector is derived from all five transverse electrodes while the Y or vertical, component of the spatial vector is derived from electrodes *H* *M* and *F* (potential difference V_y appearing between electrode *H* and a junction of two resistors joining *M* and *F*) These electrodes are connected directly

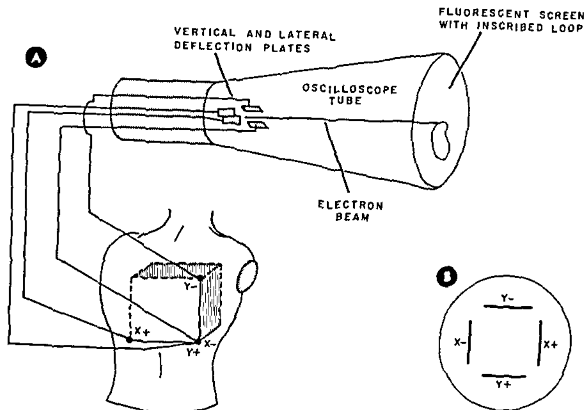
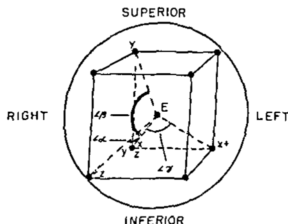


Fig 70 –Relationship between the lead electrodes and the deflection plates of the oscilloscope in the cube system of lead electrode placement **A**, electrode placement **B** polarity of deflection plates viewed from the face of the oscilloscope tube

Fig 71 –*Theoretical basis of Grishman's cube system* of lead electrode placement Note that ∠α subtended by lead Zc with *E* ∠β subtended by lead Yc *with E and* ∠γ *subtended by lead Xc with E are* equal to one another (See text for details)



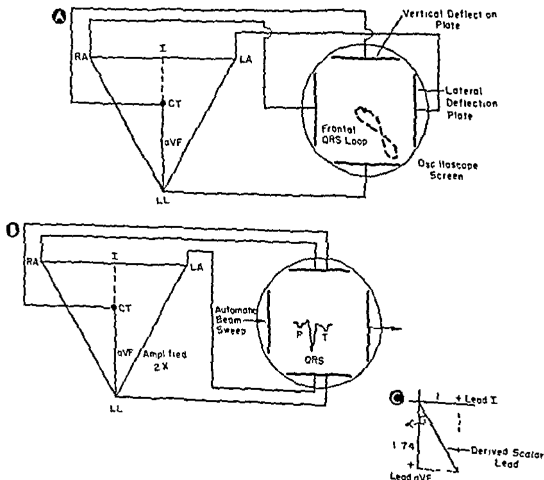
If transverse lead Xc and vertical lead Yc are attached to the two pairs of deflection plates the oscilloscope records the projection of the cardiac spatial vector on the frontal plane Similarly lead Xc and sagittal lead Zc define the projection of the vector on the horizontal plane and leads Yc and Zc the projection of the vector on *the right sagittal plane The* polarity of the deflection plates and the standardization of the component leads are the same for each planar projection in the cube system of electrode placement Consequently regardless of the plane being recorded a 1 mv input into the circuit causes the electron beam to descend at a 45 angle inferiorly and to the observer's right or the screen's left (Hereafter the direction will be stipulated in terms of the screen's left or right)

The two electrodes of each vectorcardiographic *lead are attached to a pair of* deflection plates in such a way that the relationship of the lead electrodes to the point source of potential in the body (the heart) is duplicated in the relationship *of the deflection* plates to the resting position of the electron beam

DERIVATION OF SCALAR LEAD DEFLECTIONS FROM THE VECTORCARDIOGRAM

... derive ... en ... lly to be recorded The vectorcardiographic ... ion of scalar lead deflections can be accomplished electronically or manually (or by visual inspection)

Electronic method of derivation —In this method (*Fig 74*) the axes of the component vectorcardiographic leads are treated as lead vectors having a definite magnitude or length and an effective direc-



perpendiculars dropped from the assigned lengths of these leads and second, by calculating the angle subtended by the diagonal and either of the two lead axes In this instance lead aVF was selected. Therefore

$$\tan \alpha = \frac{\text{Opposite side}}{\text{Adjacent side}} \quad \frac{1}{1\ 74}$$

$$\tan \alpha = 0.577$$

$$0\ 577 = \tan 30$$

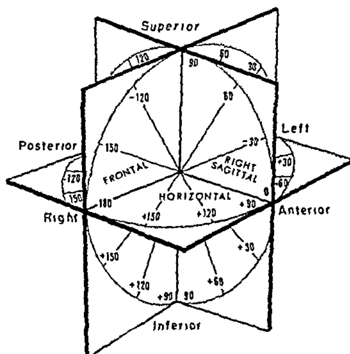


Fig 73 —Reference frames for the horizontal right

axis is situated to the left in the horizontal and frontal ref erence frames and anteriorly in the right sagittal plane

to computing and compensating networks the out puts of which are V_x V_y and V_z which are propor tional to the corresponding components of the dipole with equal standardization factors An important ad vantage claimed by advocates of the Frank system (though not all authorities agree) is its relative insen sitivity to variations in dipole position within the lim its usually encountered in clinical vectorcardiography

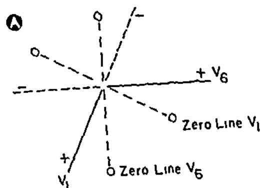
Our experience with the Frank system of recording vectorcardiograms has not been extensive as yet but our preliminary impressions of this system compared with the cube system of lead electrode placement are as follows (1) The QRS sÊ loops recorded with the two systems from the same subject generally show surprisingly close agreement in their predominant features (2) With the Frank system the QRS sÊ loops tend to have a more posterior and inferior orien tation than corresponding loops recorded from the same persons with the cube lead system In addition normal QRS sÊ loops recorded with the Frank lead system quite frequently exhibit rightward posterior and sometimes superior terminal deflections in con trast with the relative infrequency of this finding in normal QRS sÊ loops obtained with the cube lead sys tem (3) On the whole the QRS sÊ loops recorded with the Frank system show closer agreement with the electrocardiogram than loops obtained with the cube system (4) Perhaps the most striking and frequently observed difference in the vectorcardiograms recorded with the two systems of lead placement is the wider spatial angle subtended by the long axes of the QRS sÊ and T sÊ loops recorded with the Frank system in comparison with the spatial angle of the QRS sÊ–T sÊ loops obtained with the cube system The authors have not yet determined the normal range of variation of the spatial angle of the QRS sÊ and T sÊ loops in vectorcardiograms recorded with the Frank lead system

As previously indicated most of the vectorcardio grams presented in this text were recorded with the cube lead system and so the descriptions of normal and abnormal vectorcardiograms will pertain mainly to records obtained with this reference system and will apply only in a general sense to vectorcardio grams obtained by other methods of electrode place ment

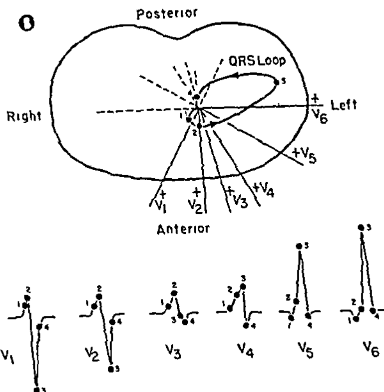
The reference frame used to indicate descriptively the orientation of the vector loops in a body plane (see Fig 73) is the same as that utilized in earlier chapters to describe the orientation of mean vectors This reference frame can be used for each body plane zero degrees in the figure lying to the left in the frontal and horizontal planes and anteriorly in the right sagit tal plane The upper half of the reference figure is marked off in −30° segments from 0° on the screen s left to 180° on the screen s right while the lower half of the figure is marked off in +30 segments clockwise from the screen s left to right Thus +90 is located inferiorly in the sagittal and frontal planes and anteriorly in the horizontal and −90° lies supe riorly in the sagittal and frontal planes and posteriorly in the horizontal For convenience and simplicity in this text the reference frames will be visualized in terms of the familiar scalar leads as shown in Table 3

TABLE 3 —PLANAR VECTORCARDIOGRAPHIC REFERENCE FRAMES EXPRESSED IN TERMS OF SCALAR LEADS

Axis	Horizontal	Right Sagittal	Frontal
0 to 180	Lead V	Lead V	Lead I
+90 to −90	Lead V_4	Lead aVF	Lead aVF
+30 to −150	Lead V_5		Lead aVR
+60 to −120	Lead V_4		Lead II
+75 to −105	Lead V		
+120 to −60	Lead V_1		Lead III
+135 to −45	Lead V_{4R}		Lead aVL
+150 to −30	Lead V_R		



Axes of Derivation of Leads V_1 and V_6



Derivation of the QRS Complexes in the Precordial Leads from the Schematic Horizontal QRS Loop

dial leads can also be derived

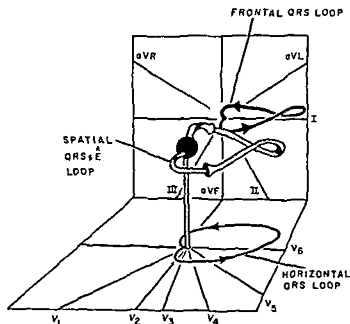
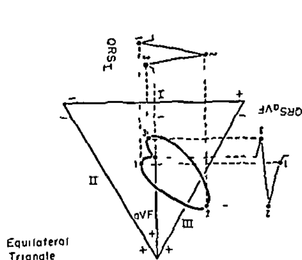
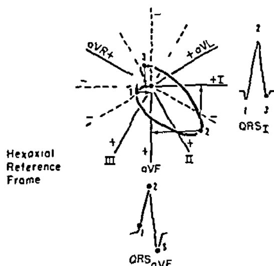


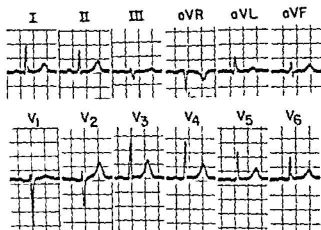
Fig 75 —Schematic diagram of QRS sÊ loop showing its projections on the frontal and horizontal reference frames formed by scalar leads I through aVF and V_1 through V_6

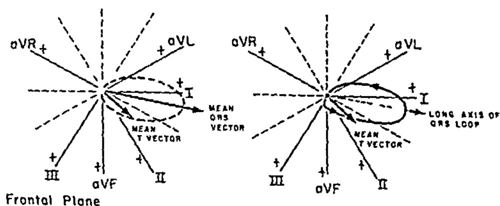




... of the QRS deflection in leads I and aVF from a schematic frontal QRS loop using the

... ous vectors drawn from the origin of the loop to the points in question ... The projection of (or electrical null point) is always superimposed on the center of the appropriate reference frame





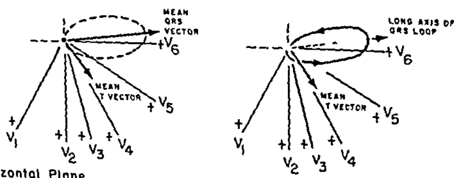


Fig 78 – Construction of vector QRS loops from the electrocardiogram. (The method is described in detail in the text)

tion As vector quantities the two lead axes can be added electronically to yield a third resultant lead vector For example if the outputs of the transverse lead Xc and vertical lead Yc (or leads I and aVF as in Fig 74) were to be added electronically and then applied to the vertical pair of oscilloscope plates the electron beam would respond to potential variations across the vertical plates as if in effect a single scalar lead intermediate between the two component leads were being recorded Moreover by amplifying one of the component leads more than the other the axis of the derived lead could then be rotated toward or away from either of the component vectorcardiographic leads Thus if the amplification of the two component vectorcardiographic leads is varied according to the appropriate amplification coefficients which have been calculated lead deflections can be derived for any scalar lead whose axis is situated in the plane defined by the component leads

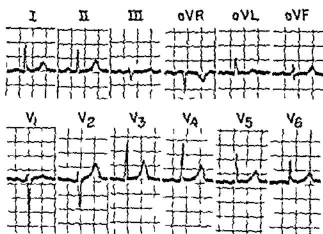
Manual method of derivation —Scalar lead deflections can also be derived manually from the vectorcardiogram but in actual practice mere inspection of the vectorcardiogram is usually sufficient to translate the vector loops into their scalar electrocardiographic equivalents As will be recalled a vectorcardiographic loop is in essence the planar projection of the pathway traced in space by the tips of all instantaneous resultant vectors generated in sequence during the P QRS or T intervals (see Fig 75) Thus a line drawn from the cardiac dipole center to any point on the QRS loop represents the planar projection of the resultant spatial vector for a given instant of the QRS interval These instantaneous vectors or the points on the loop corresponding to the termini of the vectors can be projected on any lead axis passing through the origin of the loop The positions of these projected points on the axis of the lead concerned can then be plotted on an ordinate and their times of occurrence on an abscissa to form the derived electrocardiographic deflection (Figs 76 and 77) With increased proficiency in the use of this method the scalar leads can be derived merely by inspection of the vector loop and by superimposing it mentally on the frontal horizontal or sagittal reference frames

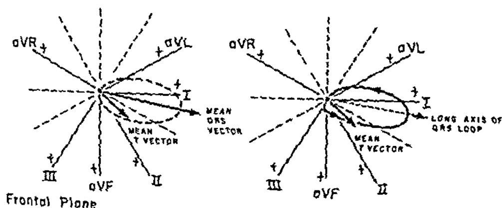
CONSTRUCTION OF VECTOR LOOPS FROM THE ELECTROCARDIOGRAM

To construct from the electrocardiogram a vector loop resembling at all closely one actually recorded by the vectorcardiograph is a formidable procedure The two scalar leads selected for the construction of the frontal or horizontal projection of the spatial vector loop must record the two components of the dipole vector in this plane The frontal plane components of a cardiac vector are the transverse (X) and vertical (Y) components and the horizontal plane components are the sagittal (Z) component and transverse components In addition the two scalar leads must be recorded simultaneously at rapid speed so that corresponding points on the same deflection in each of the two leads can be identified by their timing with reference to the onset of the deflection The voltage values for many such points are plotted on the axes of the leads and from these points perpendiculars are dropped The intersection of each pair of perpendicular lines corresponds to the tip of an instantaneous planar vector drawn from the center of the reference figure If the calculated positions of successive instantaneous vectors are connected by a line beginning and ending at the center of the planar reference frame a planar vector loop is obtained Obviously the greater the number of points on the lead deflection plotted on the lead axes the more accurate will be the construction of the vector loop

If the manual construction of vector loops from the electrocardiogram is to have any practical merit in clinical electrocardiography the method should utilize the extremity and precordial leads recorded routinely (Fig 78) For example the frontal plane vector loop would be constructed from any two of the bipolar or unipolar extremity leads and the horizontal vector loop from any two precordial leads (or from lead I and lead V_2) A number of objections can be raised to the use of the unipolar precordial leads for this purpose one of the foremost being the proximity of the exploring electrodes to the heart Moreover there is no consistent mathematical relationship between the individual precordial leads themselves and between the extremity and precordial leads And so for this reason the magnitude of the anteroposterior component (Z) of the dipole vector as determined in the precordial lead cannot be correlated quantitatively with the magnitudes of the transverse and vertical components determined from the extremity leads

The most serious shortcoming of the routine electrocardiographic leads if used to construct the vector loop is that the leads are recorded not simultaneously but sequentially This lack of phase relation





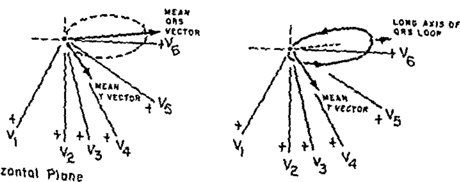


Fig 78 -Construction of vector QRS loops from the electrocardiogram. (The method is described in detail in the text)

ship in the various leads is discussed below *In short* to achieve a high degree of accuracy in the manual construction of loops from the routine electrocardiographic leads is an obvious impracticality if not an impossibility However if the inaccuracy of the method is recognized the attempt to visualize the routine electrocardiogram in terms of vector loops can be of advantage *in several respects* (1) *It tends to promote a broader understanding of the relation*ship between vectorcardiography vector projection methods of electrocardiographic analysis and routine electrocardiography (2) It represents an approach albeit an approximate one to the application of vec*torcardiographic observations and data to the inter*pretation of scalar electrocardiograms (3) It permits a more detailed and a more complete application of vector principles to the teaching of electrocardiography

The method described and used effectively by Grant and Estes to construct QRS loops is based on calculation of the mean QRS vector for each 0 02 second of the QRS interval The objection has been raised that the lack of phase relationship between consecutively recorded scalar leads precludes accurate construction of mean vectors for such short time intervals Grant and Estes answer this objection by pointing out that the mathematical relationship between the individual deflections or portions of deflections in the different extremity leads (Lead I + Lead III = Lead II and Lead aVR + Lead aVL + Lead aVF = 0) makes it possible to determine the *onset of the QRS interval without having to record* the leads simultaneously However this means of determining phase relationship has at least two limitations (1) It cannot be applied to the precordial leads since they are not mathematically interrelated nor can they be related to the extremity leads (2) As regards the extremity leads the inertia of the direct writing stylus the relatively slow paper speed routinely used and other physical limitations imposed on recording accuracy—all can lead to error in the construction of vector loops despite the use of the above formulas

Since at best calculated vector loops display *merely a rough similarity to those actually recorded* it would seem advantageous to use the simplest method of construction yielding satisfactory results The method described in this section entails *merely* (*a*) inspection of the scalar leads and (*b*) estimation of the approximate orientation of the initial deflection the maximal deflection (corresponding to the *long axis of the QRS* loop and the mean QRS vector) and the terminal deflection of the QRS loop

Method of constructing vector loops (Fig 78) – 1 The mean QRS vector for the frontal plane is determined from the bipolar limb leads and the horizontal mean QRS vector is determined *from the precor*dial leads as previously described The orientation *of these vectors roughly corresponds to that of the* maximal deflection or long axes of the frontal and horizontal QRS loops

2 The QRS loop ordinarily projects fairly symmetrically on either side of the mean QRS planar vector (corresponding to the maximal mean instanta*neous QRS vector of the QRS loop)* as one might anticipate since the lead whose axis is perpendicular to the mean vector (the transitional lead) registers an equiphasic RS deflection This implies that a perpendicular line through the midpoint of the axis of *derivation of the transitional lead bisects the planar* QRS loop into two limbs the efferent limb and the afferent limb

The *efferent limb* is the first limb of the loop to be written It is inscribed away from the point of origin of the loop and normally projects entirely on the positive half of the axis of derivation of the transitional lead

The *afferent limb* is inscribed toward the point of origin of the QRS loop and normally projects entirely on the negative half of the axis of derivation of the transitional lead

With these points in mind one can sketch the loop *in preliminary form so that it is oriented symmetrically* along the axis of the mean QRS planar vector

3 This crude QRS loop can then be altered in its initial and terminal portions (and if necessary in the efferent and afferent limbs as well) until the schematic loop conforms approximately to the relative magnitude and direction of the initial maximal and terminal components of the QRS complex in each lead *The direction of inscription* of the constructed frontal and horizontal QRS loops is often evident from the way the estimated instantaneous vectors develop but occasionally it is not possible to deduce this information from abnormal electrocardiograms particularly those showing marked alterations in ventricular depolarization such as right bundle branch block right ventricular hypertrophy and myocardial infarction

4 There is probably no advantage to be gained by construction of P and T loops The mean P and T vectors provide sufficient information

THE NORMAL VECTORCARDIOGRAM

The spatial vectorcardiogram consists of three spatial loops which appear in close succession during a single cardiac cycle The vector loops are produced by mean instantaneous P QRS and T spatial forces To indicate that these loops depict the instant-to-instant change in the direction and magnitude of the equivalent dipole (E) treated as a spatial vector (sÊ) they are designated the P sÊ QRS sÊ and T sÊ loops It is not possible to record these spatial loops directly but wire models of the loops can be constructed from their horizontal sagittal and frontal projections in the vectorcardiogram However this is seldom necessary in clinical vectorcardiography since most vectorcardiograms can be interpreted with reasonable accuracy from the planar projections themselves For this reason the vectorcardiographic descriptions given in this and subsequent chapters will deal with each planar projection more or less individually The fact should be borne in mind however that the horizontal sagittal and frontal projections of a given vectorcardiogram represent merely different views of the same spatial loop or loops (P sÊ QRS sÊ and T sÊ loops) For the sake of convenience the following terms will frequently be used hereafter in this text

Horizontal P loop —The projection of the P sÊ loop on the horizontal plane

Sagittal P loop —The projection of the P sÊ loop on the sagittal plane

Frontal P loop —The projection of the P sÊ loop on the frontal plane

(The same terminology will be applied to the *planar projections* of the QRS sÊ and T sÊ loops In more general terms the projection of a spatial loop [or loops] on a plane will sometimes be referred to as a *planar loop* [or loops])

The orientation of a planar loop will be expressed in plus or minus degrees of the appropriate reference frames However the magnitudes of the planar loops if described will be defined largely in relative or qualitative terms rather than quantitatively The reason for this is that there are too few quantitative studies of the normal and abnormal spatial vectorcardiogram available to provide any reliable standards for the magnitudes of the loops Moreover until more satisfactory lead systems are devised there seems little to be gained from attempting to quantitate vector magnitude

Although we have recorded vectorcardiograms from many normal subjects the description of the norm[illegible] series is due to the fact that we found that our preliminary results agreed with data obtained previously by other investigators with larger study series

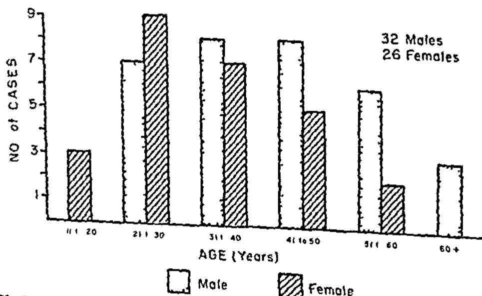
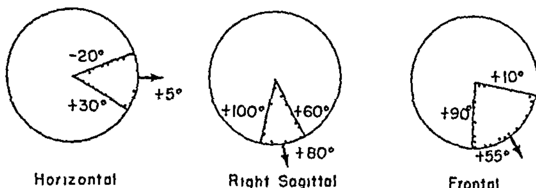
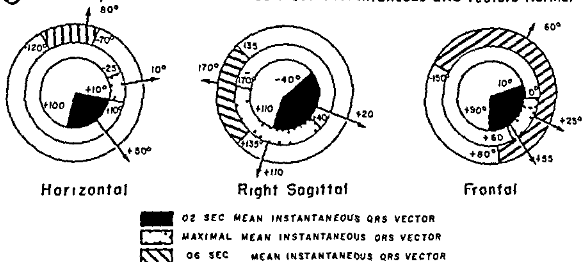


Fig 79 —S[illegible]
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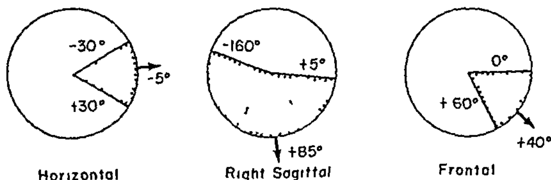
A *Maximal Mean Instantaneous P Vector — Normal*



B 02 Sec, Maximal and 06 Sec Mean Instantaneous QRS Vectors-Normal



C Maximal Mean Instantaneous T Vector — Normal



D Spatial Angle of Divergence of QRSsÊ and TsÊ Loops-Normal

% of Normal VCGs	Spatial Angle
50 %	10° or Less
35 %	11° to 30°
15 %	31° to 45°

Average Spatial Angle 17°

Fig 80 —Legend on facing page

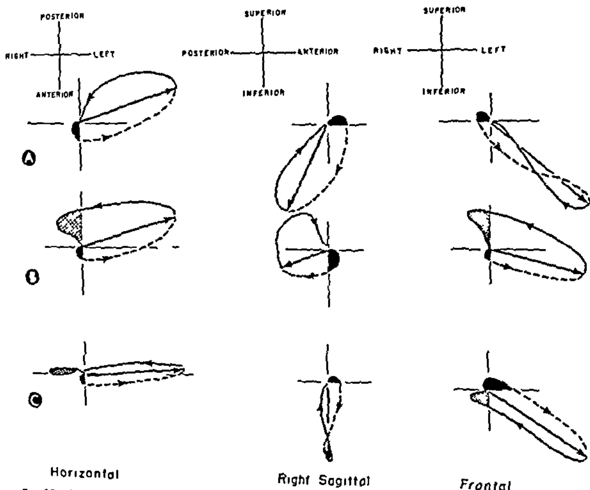


Fig 81 —A B and C the schematic planar p o ection of t ORS planar loop the solid portion of the loop correspor *sents the long axis or max m l m n in* efferent ce loop The c *QRS* loop i

F g 80 —The normal orientations of the *v*

TABLE 4 —PLANAR PROJECTIONS OF THE P sÊ LOOP OF THE NORMAL VECTORCARDIOGRAM

PLANE	ORIENTATION OF THE MAXIMAL MEAN INSTANTANEOUS VECTOR OF THE P sÊ LOOP*			DIRECTION OF INSCRIPTION	GENERAL CONFIGURATION
	Extreme Range†	Av	Usual Range†		
Horizontal	−20 to +30	+5*	0 to +5	Counter clockwise	The P sÊ loop tends to be smaller and perhaps more variable in contour in this projection than in the frontal and sagittal projections It may be triangle shaped oval or elongated or show a figure-of-eight configuration
Right sagittal	+60 to +100	+80	+80 to +90	Clockwise	The P sÊ loop in the sagittal projection is usually oval and elongated although occasionally it is triangle shaped
Frontal	+10 to +90*	+55	+20 to +60	Counter clockwise	The P sÊ loop in the frontal projection can be thin and elongated as in the sagittal projection or it can be triangle shaped

*The range in orientation as expressed in degrees by paired values is always to be read in a clockwise direction in the appropriate reference frame This convention will be adhered to throughout the text

†Extreme range = the extreme limits of the range in orientation of a given vector usual range = the range in orientation of a given vector in 85% of the cases studied

P sÊ Loop

The P sÊ loop in its planar projections has been studied very little as yet probably because it is technically the most difficult component of the vectorcardiogram to record satisfactorily The general features of the planar P loops of the vectorcardiogram as observed by us are presented in Table 4 (see also Fig 80 A)

In summary the P sÊ loop normally is oriented spatially to the left inferiorly and slightly anteriorly and is counterclockwise inscribed in the horizontal and frontal projections and clockwise inscribed in the right sagittal projection

QRS sÊ Loop

In describing the planar projections of the normal QRS spatial loop it will be necessary frequently to refer to specific portions or components of the loop In such instances the following terminology (see also Fig 81) will be used

Initial deflection —This is arbitrarily defined as the very first deflection of the QRS sÊ loop to the right and/or superiorly Normally the initial deflection is also directed anteriorly and can be considered to be produced by instantaneous forces or vectors appearing during the first 0 015 second of the QRS interval This portion of the loop reflects primarily initial left to right septal activation and subsequent beginning activation of the apicoanterior region of the right and left ventricles The initial deflection therefore corresponds roughly to the 0 01 second septal VA vector and the 0 02 second apicoanterior VA vector described in Chapter 5

Long axis or maximal vector of the QRS sÊ loop —As the term implies the longest diameter of the loop drawn from the point of origin is the long axis or maximal vector of the loop Ordinarily the terminus of the maximal vector coincides with the turning point of the loop with respect to leads I and V_6 The orientation of the maximal QRS vector in the vectorcardiogram corresponds roughly ... the 04

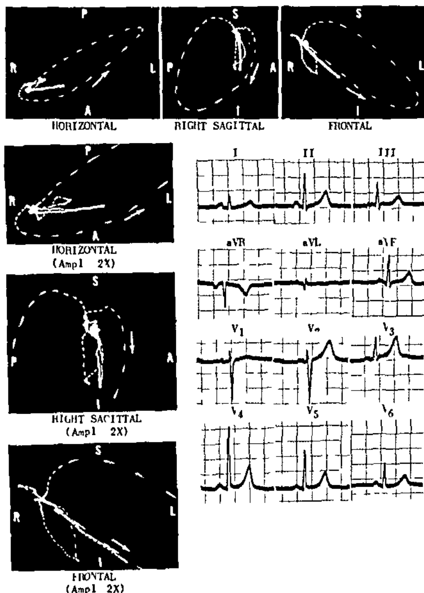
Efferent (centrifugal or outgoing) limb of the loop —The efferent limb of the QRS sÊ loop is inscribed as the electron beam moves away from the point of origin and ... of ... d

Afferent (centripetal or returning) limb —The afferent limb is inscribed as the electron beam moves back toward its point of origin It is related to activation of the lateral ...

to the last segment of the loop to be written before the loop returns to the point of origin If this portion of the loop deviates from its expected course to form a distinct

TABLE 5.—PLANAR PROJECTIONS OF THE *QRS sÊ* LOOP OF THE NORMAL VECTORCARDIOGRAM

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
0.02 second mean instantaneous QRS vector	+10 to +100	+50	+20 to +65	−40 to +110	+20	0 to +50	−10 to +90	+55	0 to +60
Maximal mean instantaneous QRS vector	−25 to +10	−10	−15 to +10	+40 to −170	+110*	+80 to +140	0 to +80	+25	0 to +40
0.06 second mean instantaneous QRS vector	−1[illegible]0 to −70	−80	−90 to −70	+135 to −135	−170	+145 to +150	−150 to +80	−60	−85 to +80
Initial deflection of QRS sÊ	Right anterior			Anterior superior or sometimes inferior			Right superior or inferior		
Direction of inscription	Invariably counterclockwise			Invariably clockwise			If the maximal instantaneous QRS vector of the frontal QRS loop lies between 0 and +40 the loop usually has a counterclockwise direction of inscription while loops with a maximal instantaneous QRS vector to the right of +40 are generally clockwisely inscribed		
Terminal instantaneous QRS vectors	Left sometimes right posterior			Posterior inferior or sometimes superior			The terminal vectors may lie superiorly in which case they tend to be located also to the right. When the terminal vectors are situated inferiorly they can be either to the right or to the left		



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normal vectorcardiograms even though its orientation is still within the limits of normal variation
and T sÊ loops in each projection tend to be concordant in orientation

deflection situated to the right and/or superiorly late in the QRS interval the term *terminal deflection or appendage* will be used.

PLANAR QRS LOOPS OF THE NORMAL VECTORCARDIOGRAM —The ranges of variation and average orientations of the 0 02 second maximal and 0 06-second mean instantaneous QRS vectors in each projection of the normal QRS sE loop are shown in Figure 80 B

HORIZONTAL QRS LOOP —Usually the normal QRS sE loop in the horizontal projection tends to be more or less oval elliptical or triangle shaped The length or long diameter of the loop is usually 1½–3 times the width (or anteroposterior dimension) In normal ch ld m nd d l ...

diminutive although identifiable and normally oriented initial deflections The efferent limb of the horizontal QRS loop usually shows a smooth (regular) anterior bowing and is inscribed in a counterclockwise direction toward the left and posteriorly On the average the maximal QRS vector or long axis of the loop tends to lie slightly posteriorly and to the left The afferent limb of the loop returns posteriorly and on the left to the point of origin and shows a smooth posterior bowing In about one third of the vectorcardiograms recorded by the authors in normal adults the terminal portion of the horizontal QRS loop extended slightly to the right and posteriorly before returning to the point of origin

RIGHT SAGITTAL QRS LOOP —The normal QRS sE loop in the sagittal projection can be oval elliptical

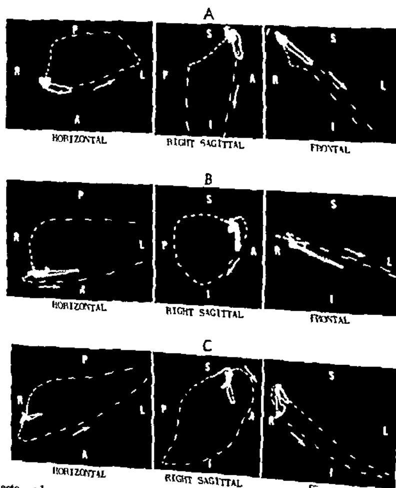


Fig 83 —A vecto ... 2 ...

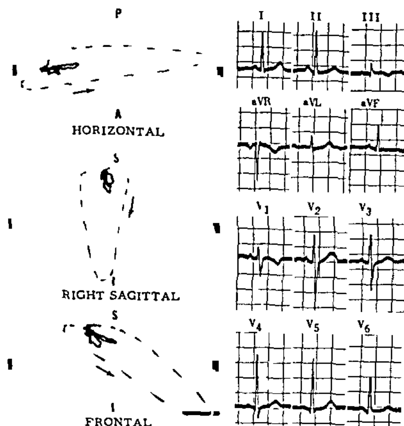
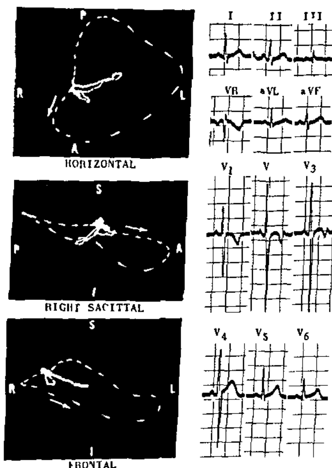


Fig 84 –Normal electrocardiogram and vectorcardiogram in a pregnant woman 28 without heart disease The *smaller of the two small loops* in each projection of the vectorcardiogram is the planar P loop the other being the planar T loop

Fig 85 –Normal electrocardiogram and vectorcardiogram in girl 18 without heart disease The tall R wave in lead V_1 of the electrocardiogram can be related to the early relatively large anterior deflection of the QRS sE loop which projects larger positive voltages on lead V_1 than are normally observed in adults These related features of the electrocardiogram and vectorcardiogram are persisting characteristics of the juvenile type of electrocardiographic and vectorcardiographic patterns The large RS deflections in the midprecordial leads of the electrocardiogram can be related to the wide anteroposterior diameter of the horizontal and sagittal QRS loops In other words the mean instantaneous QRS spatial vectors forming the efferent limb of the QRS sE loop extend far anteriorly while the vectors of the afferent limb of the loop extend even farther posteriorly the maximal positive and negative voltages being projected on those precordial leads responding primarily to the anteroposterior component of the cardiac vectors (leads V_1 through V)



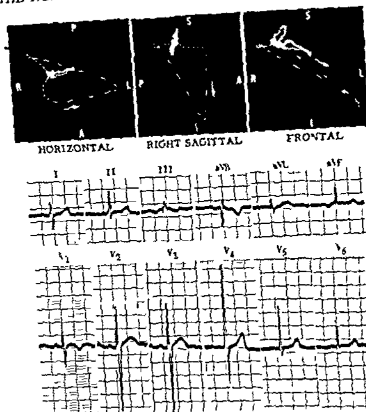


Fig 86 —Normal electrocardiogram and vectorcardiogram in boy 4 with

or triangle shaped Normally *there is an initial* deflection of the loop anteriorly and usually superiorly sometimes inferiorly The efferent limb then is written in an anterosuperior to posteroinferior direction the long axis of the sagittal loop lying inferior and slightly posterior The afferent limb subsequently returns in a clockwise direction posteriorly and inferiorly to the point of origin Occasionally the terminal returning limb extends slightly superiorly

FRONTAL QRS LOOP —In contrast with the horizontal and sagittal projections the direction of inscription of the QRS sE loop in the frontal projection can be *either* clockwise or counterclockwise depending (in part at least) on the location of the loop As a broad generalization, it can be said that *QRS sE loops* lying above the +40 axis *of the frontal* reference frame tend to be inscribed *in a counterclockwise* direction while loops situated below the +40° axis are ordinarily clockwise inscribed. In normal vectorcardiograms the frontal QRS loops observed by us were on the average located at about +25 but ranged between 0 and +60 The configuration of the frontal loop also was variable in that some loops were elongated others elliptical shaped and not infrequently loops having a figure-of-eight contour were observed. On comparing the limb leads of the electrocardiogram we noted that there was the *tendency* for the long axis of the frontal QRS loop recorded with the cube *lead system to* be situated more horizontally than the *mean frontal QRS* vector constructed from the electrocardiogram

Since the normal QRS sE *loop returns* to its point of origin an S–T *vector is not* observed *The S–T vectors* are discussed in a later section

T sÊ Loop

The normal T sE loop is usually elongated or elliptical shaped, sometimes almost linear in conf

The spatial angle* (see Table 6) between the two loops ordinarily does not exceed 60° in normal subjects (Fig 80 D) and as a general rule the same holds true for the planar angle subtended by the long axes of the QRS sÊ and T sÊ loops projected on the horizontal or frontal plane

The outgoing or efferent limb of the normal T sÊ loop is almost always inscribed more slowly than the returning limb and this is reflected in the fact that the initial limbs of the electrocardiographic T waves have more gentle slopes than the terminal limbs which either descend abruptly or rise rapidly to the base line As would be expected the direction of inscription of the T sÊ loop in each projection tends to be the same as that of the QRS sÊ loop although this can be somewhat variable Generally, the T sÊ loop in the horizontal projection is counterclockwise inscribed and in the sagittal projection clockwise inscribed while in the frontal projection the T sÊ loop can be written in either direction (Figs 82–86)

TABLE 6 –T sÊ Loop and Spatial QRS sÊ–T sÊ Angle

Orientation of the Maximal Mean Instantaneous Vector of the T sÊ Loop	Range	Average
Horizontal	−30 to +30	−5
Right sagittal	+5 to −160	+85
Frontal	0 to +60	+40
Spatial QRS sÊ–T sÊ angle* (angular divergence of long axes of QRS sÊ and T sÊ loops)	0 to 45	17

*The spatial QRS–T angles in the vectorcardiograms of the authors series of normal patients showed the following approximate percentage distribution

Percentage of vectorcardiograms	50	35	15
Spatial QRS sÊ–T sÊ angle	10 or less	11 –30	31 –45

* [illegible] is determined by means of the trigonometric table compiled by Helm and Fowler which is reproduced with the permission of these authors at the end of Chapter 6

PART II

The Abnormal Electrocardiogram and Vectorcardiogram

Ventricular Hypertrophy
General Considerations

REVIEW OF THE PERTINENT ANATOMY AND SEPTAL-VENTRICULAR ACTIVATION SEQUENCE

Interventricular Septum

THE INTERVENTRICULAR SEPTUM is situated almost parallel to the frontal plane of the body its apical portion lying slightly anterior and inferior to its basal portion The thickness of the septal muscle mass approximates that of the basal wall of the left ventricle Both septum and free wall of the left ventricle decrease in th ckne n t

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sides of the septum the left division branches early and profusely but the right does not branch until it reaches the vicinity of the base of the anterior papillary muscle The left bundle branch distributes the excitation impulse to those regions of the muscular septum derived from the left ventricle the septal muscle contributed by the right ventricle being activated via the right bundle branch For all intents and purposes the right basal region of the septum is the only electronically significant contribution of the right ventricle to the septal muscle mass while the major portion of the septal muscle is depolarized in a left posterior to right anterior direction via ramifications of the left bundle branch Since conduction via the Purkinje fibers akes place very rapidly activation of the septal musculature despite the latter s thickness normally produces electrical forces of small magnitude As will be seen later when septal activation occurs in an abnormal fashion it is capable of generating potentials of considerable magnitude In any event with the single exception of the right basal portion of the septum which is derived from the right ventricle the remainder of the septal muscle can be considered to form the anteromedial wall of the left ventricle The significance of the foregoing facts will become evident in later discussions

Free Walls of Left and Right Ventricles

The *effective sites* or electrical locations of the right and left ventricles are determined by various factors—for example the anatomic location of the ventricles the sequence of ventricular activation and the relative magnitudes of the potentials generated in the two ventricles The effective site of the left ventricle is posterior to the left and superior the effective site of the right ventricle is to the right anterior and inferior or superior The free wall of the right ventricle is relatively thinner than that of the left ventricle and the right ventricle itself is shorter than the left The apical or trabecular walls of both right and left ventricles are thinner and the basal walls of the two ventricles thicker than other portions of the free walls and there also tends to be a parallel relationship between the magnitudes of the electrical forces generated in basal and trabecular walls The fact that the trabecular portions of the two ventricles give rise to forces of small magnitude can perhaps be attributed to another factor in addition to the thinness of the trabecular muscle and that is the deeper penetration of the Purkinje fibers into the apical myocardium Thus activation of the inner muscle occurs very rapidly via the Purkinje fibers and consequently generates negligible forces Only later when a

bounded wave front is formed during activation of outer layers of apical muscle are there produced electrical forces of significant magnitude

The hypothetical instantaneous VA (ventricular activation) vectors previously used to schematize ventricular activation in the normal heart will serve the same function in subsequent discussions of the activation process in ventricular hypertrophy bundle branch block and myocardial infarction The magnitude of these vectors normally *increases until the* maximal VA vector appears at about 0 04 second after onset of ventricular activation and tends to decrease thereafter The normal orientation timing and significance of each of these vectors were described in detail in Chapter 5 to which the reader is referred Let it suffice to say that normally the *instantaneous* VA vectors reflect predominantly the electrical effects of left ventricular activation since the activation forces generated by the right ventricle are comparatively *small*

ELECTRICAL EFFECTS OF VENTRICULAR HYPERTROPHY

Pathologically the basic abnormality of the heart muscle in ventricular hypertrophy consists of an increased cross sectional area of the individual muscle fibers without any change in their total number The altered morphology of the hypertrophied muscle *fibers can have electrical effects which lead to recog*nizable changes in the electrocardiogram although this is not an invariable rule The three general electrical effects of ventricular hypertrophy are (1) increased magnitude and rotation of the mean instantaneous QRS spatial vectors toward the effective electrical site of the hypertrophied ventricle (2) lengthening of the time required for activation of the hypertrophied ventricle and (3) rotation of the mean instantaneous T spatial vectors *away from the* mean instantaneous *QRS spatial vectors The mech*anisms by which anatomic ventricular hypertrophy *is translated* into the foregoing electrical effects are depicted *in* Figure 87 and discussed in the following paragraphs

OF THE HYPERTROPHIED VENTRICLE

It will be recalled that each instantaneous QRS spatial vector produced during simultaneous activa*tion of the ventricular free walls can be considered to* be the resultant of two component vectors which represent the electrical forces generated *in opposing* walls of the left and right ventricles (Fig 88) If the electrical forces produced by the left ventricle increase in magnitude without any change in the magnitude of the right *ventricular forces* then obviously the resultant of the left and *right ventricular forces* (the mean instantaneous QRS spatial vector) not only lengthens but also assumes more nearly the same *direction as the* left ventricular component vector Conversely if the magnitude of the right ventricular forces increases while the left ventricular forces remain unchanged the mean instantaneous QRS spatial vector rotates in the direction of the right ventricular component vector and then becomes larger

*It is evident therefore that in ventricular hyper*trophy the larger forces arising in the affected ventricle can lead both to an increased magnitude of the mean instantaneous QRS spatial vector and to rotation of the vector toward the effective site of the hypertrophied ventricle Some of the factors responsible for the increased magnitude of the forces generated by the hypertrophied ventricle are the following

1 When the cross sectional area of a muscle fiber increases *the fibers internal resistance to current flow* decreases (*Fig* 89) According to the circuit equation $I = e/(R + r)$ the current flow (I) in any closed circuit equals the electromotive force (e) of the current source divided by the sum of the resistances in the external (R) and internal (r) circuits If the electromotive force of the membrane current of a heart muscle cell is assumed to be constant it follows that a decrease in the internal resistance (r) must necessarily result in an increased *current flow in the external circuit in the conducting* medium surrounding the cell The increased current flow is transmitted to the galvanometer and produces a larger *voltage drop across its terminals*

2 As the result of the increased breadth of the muscle fibers in the hypertrophied ventricle the surface area and the thickness of the ventricular wall are increased and therefore the *hypertrophied ventricle* produces QRS forces of greater magnitude In addition for reasons to be explained below the increased thickness of the ventricular wall may result in tangential rather than radial spread of the activation wave through the involved muscle and *this gives rise* to larger QRS forces

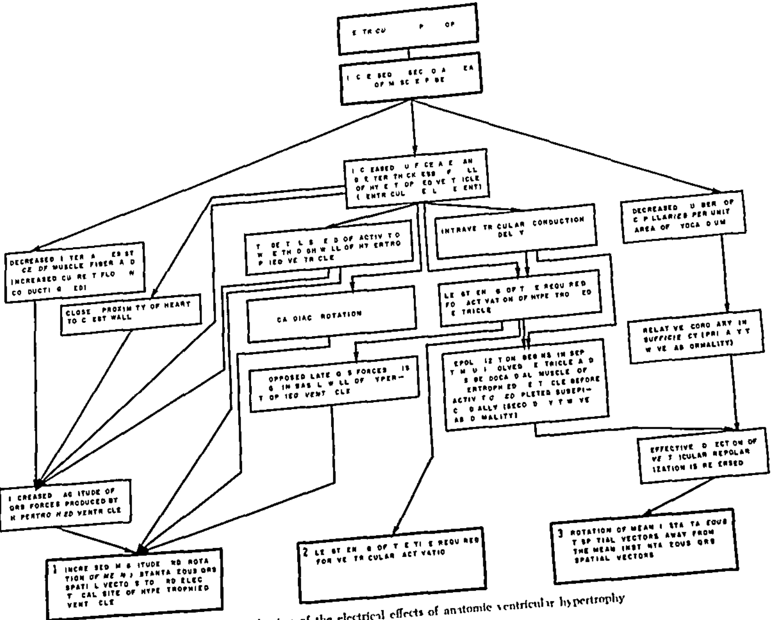


Fig 87 —Mechanism of the electrical effects of anatomic ventricular hypertrophy

and to the right superiorly and either anteriorly or slightly posteriorly in right ventricular hypertrophy

2 The mean instantaneous spatial vectors produced relatively late in the QRS interval may be rotated from their usual position owing to the fact that late QRS forces arising in the thick basal wall of the hypertrophied ventricle are unopposed by potentials from the other ventricle Such an abnormality can be attributed to the increased thickness of the wall of the hypertrophied ventricle which results in the lengthening of the time required for completion of activation in the affected ventricle This factor occasionally in conjunction with a co existing intraventricular conduction disturbance may result in activation being completed in the unaffected ventricle before basal regions of the hypertrophied ventricle have completed activation Thus late QRS forces are produced in the thickest portion of the hypertrophied ventricle and are virtually unopposed The late instantaneous vectors may represent primarily forces arising in the basal wall of the hypertrophied ventricle

3 Some authorities believe that anatomic cardiac rotation is one of the factors responsible for the displacement of the instantaneous QRS spatial vectors toward the effective electrical site of the hypertrophied ventricle For example

a) According to some authorities the mechanical effect of right ventricular hypertrophy may lead to extreme clockwise rotation of the heart on its longitudinal axis so that the left and right ventricles exchange positions Thus in right ventricular hypertrophy the instantaneous QRS vectors are oriented just as normally toward the electrically dominant left ventricle but the left ventricle is located anteriorly and to the right This explanation of the rightward and anterior orientation of the instantaneous QRS vectors in right ventricular hypertrophy has been severely criticized

b) The posterior superior and leftward orientation of the instantaneous QRS vectors in left ventricular hypertrophy has been attributed to counterclockwise rotation of the heart on its longitudinal axis In several recent studies no consistent correlation could be demonstrated between anatomic heart position and orientation of the mean QRS spatial vector or the long axis of the vectorcardiographic QRS sÊ loop It would seem therefore that in left ventricular hypertrophy cardiac rotation is not the primary mechanism responsible for the characteristic orientation of the instantaneous vectors

Lengthening of the Time Required for Activation of the Hypertrophied Ventricle

Several different mechanisms jointly or individually may be responsible for the lengthening of the time required for ventricular activation They are as follows

1 *Because the wall of the hypertrophied ventricle* is thicker than normal the distance the activation wave must travel to reach the epicardium is increased Thus onset of the intrinsicoid deflection or the appearance of the maximal vector in a lead facing the hypertrophied ventricle is delayed The greater muscle mass of the hypertrophied ventricle also causes an overall lengthening of the time required for ventricular activation as evidenced by an increased duration of the QRS interval

2 Another factor which has been implicated by some investigators is lengthening of the conduction time required for spread of excitation over the endocardium as the result of ventricular dilatation and hypertrophy

3 A localized conduction disturbance such as incomplete bundle branch block, may also contribute to the prolonged QRS interval The conduction defect can result from coronary artery disease or from compression of blood vessels supplying the subendocardium owing to increased intraventricular pressure

Rotation of the Mean Instantaneous T Spatial Vectors Away from the Mean Instantaneous QRS Spatial Vectors

The mechanisms in ventricular hypertrophy which cause the mean instantaneous T spatial vectors to diverge from the corresponding QRS vectors are described below according to the type of T wave abnormality produced in the electrocardiogram whether secondary or primary As will be recalled an abnormally wide divergence of the instantaneous QRS and T vectors which produces secondary T wave changes is not associated with an abnormal ventricular gradient since it results from a primary change in the depolarization process When the displacement of the instantaneous T spatial vectors produces primary T wave abnormalities the ventricular gradient is abnormal Consequently the underlying mechanism must entail some change in the process of recovery in the hypertrophied ventricle

Secondary abnormality of the mean instantaneous T spatial vectors—Because of the increased thickness of the ventricular wall a longer time is required for

the activation wave to pass from the endocardium to the epicardium Therefore repolarization begins in the endocardium before epicardial depolarization has been completed and the direction in which repolarization spreads thereafter is the reverse of normal Since the hypertrophied ventricle tends to be electrically preponderant during both ventricular depolarization and repolarization the reversed order of recovery in the layers of the hypertrophied ventricular wall affects the over all balance of T forces As a result the instantaneous T vectors rotate away from the instantaneous QRS vectors causing the QRS deflections and the accompanying T waves in a given lead to be oppositely directed Because of the overlapping of the depolarization and repolarization processes in ventricular hypertrophy the larger T forces produced by the hypertrophied ventricle manifest themselves early in the S–T interval by displacing the base line of the electrocardiogram The effect of these early repolarization forces can be visualized as an *S–T vector* which for obvious reasons parallels the instantaneous T vectors in the absence of true injury S–T vectors Thus scalar leads showing inverted T waves record depressed S–T segments while leads with upright T waves show S–T segment elevation Secondary ST–T wave changes of this type presumably result from the altered ventricular activation process in ventricular hypertrophy

Primary abnormality of the mean instantaneous T spatial vectors.—This type of repolarization abnormality as it occurs in ventricular hypertrophy is usually due to myocardial ischemia The latter condition can result from coronary artery disease or possibly from *relative coronary insufficiency* The concept of relative coronary insufficiency rests on the fact that ventricular hypertrophy leads to a decrease in the number of capillaries per unit volume of myocardium Moreover the cross sectional area of the muscle fibers is increased so that the inside of each cell becomes further removed from the capillaries As a result the interchange of oxygen and metabolites is impeded and ischemia ensues

Myocardial ischemia prolongs the recovery process in all layers of ventricular wall and so repolarization begins at the endocardial surface and proceeds in the *same direction as depolarization* The instantaneous T vectors swing away from the effective region of ischemia and since this usually coincides with the site of the hypertrophied ventricle the angular divergence of the QRS and T vectors becomes abnormally wide (Myocardial ischemia will be discussed in more detail in Chapter 18)

The ECG in Ventricular Hypertrophy

Although the electrocardiographic findings in ventricular hypertrophy are described in full detail in the individual sections dealing with hypertrophy of one or the other ventricle it is appropriate to conclude the introductory comments by relating the specific electrical effects of left and right ventricular hypertrophy to the electrocardiogram (see also Fig 87) under the following headings

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LEFT VENTRICULAR HYPERTROPHY

In left ventricular hypertrophy the *mean instantaneous* QRS spatial vectors of increased magn n de

m n n

RIGHT VENTRICULAR HYPERTROPHY

In right ventricular hypertrophy the mean instantaneous QRS spatial vectors of incr nd —

Au or v — u

Thus the electrocardiogram shows the following *general* abnormalities

1 Leads I and aVL record tall upright R waves and leads II III and aVF record rS deflections The limb leads usually show left axis deviation of the mean manifest electrical axis of QRS

1 Leads I and aVL recor r rs —
leads II II
aVR displa
R wave T
mean mani

and to the right superiorly and either anteriorly or slightly posteriorly in right ventricular hypertrophy

2 The mean instantaneous spatial vectors produced relatively late in the QRS interval may be rotated from their usual position owing to the fact that late QRS forces arising in the thick basal wall of the hypertrophied ventricle are unopposed by potentials from the other ventricle Such an abnormality can be attributed to the increased thickness of the wall of the hypertrophied ventricle which results in the lengthening of the time required for completion of activation in the affected ventricle This factor occasionally in conjunction with a co existing intraventricular conduction disturbance may result in activation being completed in the unaffected ventricle before basal regions of the hypertrophied ventricle have completed activation Thus late QRS forces are produced in the thickest portion of the hypertrophied ventricle and are virtually unopposed The late instantaneous vectors may represent primarily forces arising in the basal wall of the hypertrophied ventricle

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Rotation of the Mean Instantaneous T Spatial Vectors Away from the Mean Instantaneous QRS Spatial Vectors

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Secondary abnormality of the mean instantaneous T spatial vectors—Because of the increased thickness of the ventricular wall a longer time is required for

Left Ventricular Hypertrophy

NORMALLY THE LEFT VENTRICLE contributes approximately three quarters of the potentials which produce the QRS deflection With hypertrophy of the left ventricle this electrical predominance of left over right ventricle becomes all the more accentuated The greater the disparity between left and right ventricular forces the more closely the electrocardiogram resembles a levocardiogram in that the mean instantaneous QRS vectors more *nearly represent pure left* ventricular forces Thus the mean instantaneous QRS spatial vectors in left ventricular hypertrophy tend to increase in magnitude as they develop in an abnormally posterior superior and leftward direction

The ventricular activation sequence will be schematized below in terms of hypothetical instantaneous VA (ventricular activation) vectors which represent a synthesis of the findings in a typical case of left ventricular hypertrophy (see also Fig 90) The de*scription of the VA vectors in left ventricular hyper*trophy to be given in the following paragraphs should be compared with the earlier description of the instantaneous VA vectors in the typical normal heart (pp 54–59) These VA vectors in ...
heart re
vectore-
trophy

THE INSTANTANEOUS VA VECTORS

0 01 SECOND SEPTAL VA VECTOR

In left ventricular hypertrophy the septal VA vector is usually relatively normal in direction and magnitude however sometimes it has a larger magnitude than normal and extends farther anteriorly inferiorly and to the right. The greater size of the 0 01 second VA vector *possibly is a reflection* of septal hypertrophy The septum anatomically is a part of the left ventricle and can therefore *participate in the* hypertrophy process just like the ventricular free wall In other cases of left ventricular hypertrophy the 0 01 second VA vector *may be oriented* in a more left-to-right direction and less anteriorly This has been attributed, by some investigators to counter clockwise rotation of the heart on *its longitudinal* axis

VARIATIONS IN THE 0 01 SECOND SEPTAL VA VECTOR —The three types of variations follow

1 There may be normal direction and magnitude of the vector with the following results in the electrocardiogram

Leads I and V_6 —The vector projects small Q waves on these leads just as normally

Leads III aVF and V_1 —On these leads the vector projects the initial part of a small R wave

2 There may be increased magnitude but normal direction of the vector so that the following deflections are written

Leads I and V_6 —The vector projects deep but narrow Q waves on these leads

Leads III aVF and V_1 —The vector projects the initial portion of a relatively tall R wave on these leads Ordinarily *however the R/S* amplitude ratio in lead V_1 merely approaches but does not equal 1 the S wave remaining the larger component of the RS deflection *in this lead*

3 The vector may be oriented more to the right and less anteriorly

Leads I and V —The vector may ...
leads Q ...

... y because

2 Leads V_1 through V_3 or V_4 display large rS deflections and leads V_5 and V_6 tall R waves The transition point for the QRS deflection tends to be shifted to the left of its usual position near the electrode position of lead V_3 or V_4

2 Leads V_{3R} through V_2 or V_3 record QRS deflections of varying configuration (e g rR qR rsR etc) whose net voltage is positive while precordial leads to the left record QRS deflections of diminishing positive and increasing negative voltage There is therefore a reversal in the precordial QRS transition

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Lengthening of the time required for activation of the hypertrophied ventricle —The prolonged ventricular activation time of the hypertrophied ventricle causes

LEFT VENTRICULAR HYPERTROPHY

1 Delayed onset of the intrinsicoid deflection in lead V_6

RIGHT VENTRICULAR HYPERTROPHY

1 Delayed onset of the intrinsicoid deflection in lead V_1

2 An over all lengthening of the time required for activation of the ventricle so that the QRS interval is often prolonged to 0 11 or 0 12 second

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Rotation of the mean instantaneous T spatial vectors away from the mean instantaneous QRS spatial vectors —*The greater angular divergence of the mean instantaneous QRS and T spatial vectors* means that the QRS deflections and T waves in any lead tend to be directed oppositely while normally they usually have the same direction The resulting findings are as follows

LEFT VENTRICULAR HYPERTROPHY

Leads I aVL V_5 and V_6 record inverted T waves and leads II III aVF and V_1 through V_3 or V_4 record upright T waves

RIGHT VENTRICULAR HYPERTROPHY

Leads II III aVF and V_{3R} through V_3 or V_4 record inverted T waves and leads I aVL V_5 and V_6 register upright T waves

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Left Ventricular Hypertrophy

Normally the left ventricle contributes approximately three quarters of the potentials which produce the QRS deflection With hypertrophy of the left ventricle this electrical predominance of left over [illegible] The

[illegible]

sembles a *levocardiogram* in that the mean instantaneous QRS vectors more nearly represent pure left ventricular forces Thus *the mean instantaneous QRS spatial vectors in left ventricular hypertrophy tend to increase in magnitude as they develop in an abnormally posterior superior and leftward direction*

The ventricular activation sequence will be schematized below in terms of hypothetical instantaneous VA (ventricular activation) vectors which represent a synthesis of the findings in a typical case of left ventricular hypertrophy (see also Fig 90) The description of the VA vectors in left ventricular hypertrophy to be given in the following paragraphs should be compared with the earlier description of the instantaneous VA vectors in the typical normal heart (pp 54–59) These VA vectors in the typical normal heart resemble closely those actually recorded in the vectorcardiographic pattern of left ventricular hypertrophy

THE INSTANTANEOUS VA VECTORS

0 01 Second Septal VA Vector

In left ventricular hypertrophy the septal VA vector is usually *relatively normal* in direction and magnitude however sometimes it has a *larger magnitude* than normal and extends *farther anteriorly* inferiorly and to the right. The greater size of the 0 01 *second VA vector possibly* is a reflection of septal hypertrophy The septum *anatomically is a part of the left ventricle* and can therefore participate *in* the hypertrophy process just like the ventricular free wall In other cases of left ventricular hypertrophy *the 0 01 second VA* vector may be oriented in a more left *to-right* direction and less anteriorly This has been attributed by some investigators to counterclockwise *rotation* of the heart on its long*itudinal axis*

Variations in the 0 01-second septal VA vector.—The *three* types of variations follow

1 There may be normal direction and magnitude of the vector with the following results in the electrocardiogram

Leads I and V_6 —The vector projects small Q waves on these leads just as normally

Leads III aVF and V —On these leads the vector projects the initial part of a small R wave

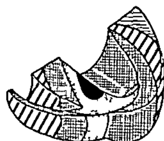
2 There may be increased magnitude but normal direction of the vector so that the following deflections are written

Leads I and V_6 —The vector projects deep but narrow Q waves on these leads

Leads III aVF and V —The vector projects the initial portion of a relatively tall R wave on these leads Ordinarily however the R/S amplitude ratio *in lead* V_1 merely approaches but does not equal 1 the S wave *remaining* the larger component of the RS deflection in this lead

3 The vector may be oriented more to the *right* and less anteriorly

Leads I and V_6 —The vector may project somewhat deeper Q waves than normal on leads I and V_6 These Q waves are usually not particularly striking, in contrast with the preceding variant probably because



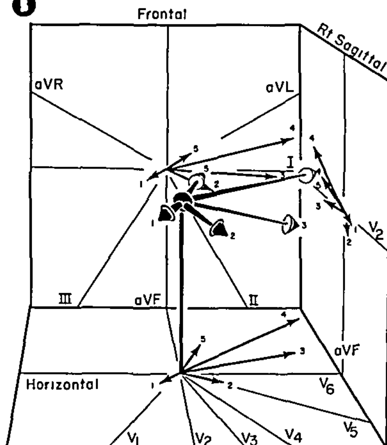


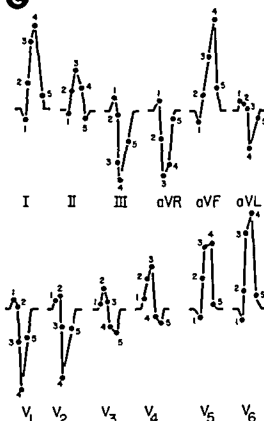












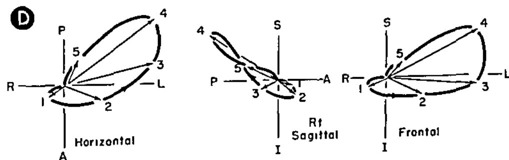


Fig 90 —Instantaneous VA vectors in left ventricular hypertrophy (LVH) A sequence of septal ventricular activation B the instantaneous VA spatial vectors These vectors are numbered so that they can be related in an approximate

the R waves in leads I and V_6 are larger than normal and so the relative (though not the absolute) *size* of the Q and R waves in these leads may differ little from normal

Leads III aVF and V_1 —The more rightward and the less anterior and inferior the orientation of the vector the less is its projection on the lead axes of leads III aVF and V_1 A smaller anterior component

trophy (see below)

0 02 Second Apicoanterior VA Vector

In left ventricular hypertrophy the marked electrical predominance of the left ventricle can be manifested as early as 0 02 *second after onset of the QRS* interval In this event the apicoanterior VA vector is larger than normal and directed more to the left and either less anteriorly and inferiorly or somewhat posteriorly or superiorly Just as was the case with the 0 01 second septal VA vector and for the same reason the 0 02 second vector *sometimes may* have an exaggerated anterior and rightward projection

1 The initial septal VA vector may be oriented more to the right and less anteriorly and inferiorly than normal and the 0 02 second apicoanterior VA vector more to the left posteriorly and superiorly

Leads I and V —On these leads the vectors project a Q wave and the upstroke of an R wave both deflections being larger than normal but of normal relative proportions

Leads III aVF and V —Very small initial R waves are often produced In some cases lead III displays a Q wave and lead V_1 a QS deflection

2 *Both* the 0 01 and 0 02 *second* VA vectors may have an exaggerated *anterior* rightwar[illegible]

by

gr[illegible] above

Leads III aVF and V —R waves which *are* taller than normal but of normal duration Lead V_1 is particularly likely to show this finding

No satisfactory explanation has as yet been offered for the fact that initial R waves may be absent in lead V_1 in 10–30% of cases of left ventricular hypertrophy and in lead V in 1–4% of the cases In all probability antecedent anteroseptal myocardial infarction is *responsible* for this finding in *some cases* but *certainly not in all*

0 04 Second Left Ventricular VA Vector

This vector reflects the accentuated electrical preponderance of the left ventricle in left ventricular hypertrophy more clearly than do the preceding vectors Thus the magnitude of the 0 04 second VA vector in left ventricular hypertrophy is greater than normal and the orientation of the vector more posterior and superior and to the left

Leads I and V_6 —The projection of this vector on the axes of leads I and V_6 contributes to the tall R waves inscribed in these leads

Leads III aVF and V_1 —The vector projects deep S waves on leads V_1 and V_2 and smaller S waves on leads III and aVF

Despite the greater magnitude of the 0 04 second VA vector in left ventricular hypertrophy it is *not* the maximal vector to appear during ventricular activation The maximal vector appears 0 01–0 02 second later the delay possibly being the result of either or both of the following factors (1) the over all lengthening of the time required for ventricular activation owing to the marked thickening of the left ventricular wall and (2) *the relatively greater hypertrophy* of the basal and posterolateral walls of *the left* ventricle These regions are activated late in the QRS interval normally Inasmuch as *they generate the maximal* forces in left ventricular hypertrophy and may be activated even later than in the normal heart the maximal vector is also delayed in onset

0 06-Second Basal VA Vector

The abnormal magnitude and orientation of the 0 06 second VA vector *are indicative of the delayed* onset of the maximal ventricular activation forces in left ventricul[illegible]

vector can be visualized as extending farther to the left and lying more posterior and superior than all the foregoing VA vectors The basal VA vector tends to parallel the negative portion of the lead V_1 axis and may project maximal positivity on lead V_6

Leads I and V_6 —This vector coincides approximately with the peak of the tall R waves in lead V_6 and contributes to the tall R wave registered in lead I

Leads III aVF and V_1 —The vector coincides approximately with the nadir of the S wave in leads III AVF and V_1

Since the peak of the R wave in lead V_6 corresponds to the time of onset of the intrinsicoid deflection in this lead it is evident that the fact that the 0 06 second VA vector is the maximal VA vector to appear during ventricular activation (rather than the 0 04 second VA vector as is normally the case) means that there is a delayed onset of the intrinsicoid deflection in lead V_6 On the other hand onset of the intrinsicoid deflection occurs at the normal time in lead V_1

As will be recalled the maximal instantaneous VA (or QRS) vector in the horizontal plane corresponds roughly to the horizontal mean QRS vector calculated from the precordial leads of the electrocardiogram The orientation of the mean QRS vector is established by identifying the precordial lead which registers the equiphasic or transitional RS deflection the so called *transitional lead* Precordial leads located to the right of the transitional lead register low R waves and deep S waves in left ventricular hypertrophy since they lie in the negative field of the mean QRS vector leads to the left of the transitional lead record tall R waves because they are situated in the positive field of the vector The marked posterior orientation of the instantaneous vectors and horizontal mean vector which characterizes left ventricular hypertrophy is responsible for the fact that the transitional or equiphasic RS deflection frequently is recorded by a lead situated to the left of the normal transitional zone between leads V_3 and V_4 If for example the horizontal mean QRS vector lies between −60 and −90° in the horizontal reference frame a lead intermediate between leads V_5 and V_6 would record the transitional deflection leads V_1 through V_5 displaying resultantly negative or downwardly directed QRS deflections This is the conventional electrocardiographic pattern of clockwise rotation However when there is marked clockwise rotation" with RS deflections in lead V_5 or V_6 the QRS voltage in the latter leads may not meet the criteria for voltage of left ventricular hypertrophy

0 08 Second Terminal VA Vector

For a prolonged interval after completion of right ventricular activation basal portions of the left ventricle continue to depolarize Accordingly the QRS interval in left ventricular hypertrophy is usually prolonged sometimes to 0 12 second and the terminal VA vector appears later than normal Since the forces produced by activation of basal portions of the left ventricle are unopposed by forces arising elsewhere the terminal VA vector is directed even farther posteriorly but less superiorly than the VA vectors preceding it

Leads I and V_6 —This vector projects diminishing positivity on leads I and V_6

Leads III aVF and V_1 —On these leads the vector projects diminishing negativity

VENTRICULAR REPOLARIZATION

Before depolarization in the basal regions of the left ventricle is completed repolarization begins at the endocardial surface of the left ventricle (see Chapter 8) In the right ventricle recovery begins at the epicardial surface just as normally although the resulting T forces are negligible compared to the repolarization forces arising in the left ventricle The reversal in the direction of repolarization in the electrically dominant left ventricle causes the instantaneous T vectors to rotate away from the instantaneous VA vectors Thus electrocardiographic leads registering upright QRS deflections usually record inverted T waves (leads I aVL V_5 and V_6) while leads displaying deep S waves have upright T waves (leads V_1 and V_2) Because of the overlapping of the depolarization and repolarization processes repolarization forces begin to act on the electrocardiograph before the QRS forces have subsided Since these repolarization forces which appear early in the S–T interval are identical with the forces producing the T wave and have the same direction the S–T segments tend to be depressed in leads recording inverted T waves and slightly elevated in leads registering upright T waves Sometimes the QRS deflections and T waves in left ventricular hypertrophy are concordant in such cases the authors phrase the interpretations ? left ventricular hypertrophy on the basis of high QRS voltage alone (to be discussed later)

GENERAL ECG FINDINGS AND RELATED DIAGNOSTIC CRITERIA IN LEFT VENTRICULAR HYPERTROPHY

Increased magnitude and a more posterior superior and leftward orientation of the mean instantaneous QRS spatial vectors

EXTREMITY LEADS

1 Leads I and aVL record tall upright QRS deflections while leads II III and aVF display rS deflections The S wave in lead III is usually deep

PRECORDIAL LEADS

1 Leads V through V_4 or V_4 record small initial R waves followed by very deep S waves while leads V_4 and/or V register small Q waves followed by very tall R waves

QRS Voltage Criteria

R + S_{III} ≧ 25 mm.

R aVL > 11 mm

R aVF > 20 mm

S , ≧ 24mm

R , or R_{V_6} > 26 mm.

R , or R , + S , > 35 mm

~ There is often left-axis deviation of the mean manifest electrical axis of QRS (Â QRS) When ...

2 Since the mean instantaneous QRS spatial vectors ...

... this point) is often a ...

Orientation of Â QRS (Fig 9 1 A)

	Frontal Plane		Horizontal Plane
Range	−60 to +90	Range	−60 to 0
Average	+ ,	Average	−25

Over-all lengthening of the time required for ventricular activation

EXTREMITY LEADS

PRECORDIAL LEADS

Criteria

Increased QRS duration 0 11–0 12 second

Delayed onset of the intrinsicoid deflection in lead V 0 05 second or later after onset of the QRS interval Normal time of onset of intrinsicoid deflection in lead V ,

Rotation of the mean instantaneous T spatial vectors to the right anteriorly and inferiorly away from the mean instantaneous QRS spatial vectors

EXTREMITY LEADS

PRECORDIAL LEADS

Criteria

1 Lead aVL or aVF 0 5 mm or more depression of S–T segment (representing early abnormally directed T wave forces)

2 Lead aVL or aVF Flat, diphasic or inverted T wave (with an R wave of 6 mm. or more amplitude) plus 0 5 mm. or more depression of S–T segment

3 Lead aVR Upright T wave

1 Lead V or V S–T segment depression and/or low or inverted T waves

ORIENTATION OF A T (FIG 91 A)

Frontal Plane		*Horizontal Plane*	
Range	+10 *to* −140	Range	+45 to +170
Average	+120	Average	+135

Comment The transition from upright T waves and elevated S–T segments to inverted T waves and depressed S–T segments usually occurs just to the left or right of or in the same lead as that recording the transitional equiphasic RS deflection However in the presence of digitalis effect or of superimposed myocardial ischemia precordial leads to the right of the transition point for the QRS *may record inverted T waves*

LIMITATIONS IN DIAGNOSTIC SPECIFICITY OF THE CRITERIA IN LEFT VENTRICULAR HYPERTROPHY

The diagnostic accuracy of the electrocardiogram in left ventricular hypertrophy leaves much to be desired In the first place left ventricular hypertrophy merely exaggerates the normal electrical preponderance of the left ventricle *without otherwise causing* any marked disturbance in the orientation of the mean *instantaneous QRS spatial vectors For this reason the configuration of the QRS* deflections in left ventricular hypertrophy aside from the large voltage is not particularly distinctive while abnormalities of the QRS configuration usually figure prominently in the diagnosis of conditions such as right ventricular hypertrophy bundle branch block and myocardial infarction Since characteristic changes in QRS configuration are lacking in left ventricular hypertrophy the electrocardiographic diagnosis must therefore depend on other criteria—namely QRS voltage orientation of the planar mean QRS vectors the QRS duration and pre intrinsicoid deflection time and finally the angular divergence of the QRS and T forces Each of these categories of diagnostic criteria deserves critical *scrutiny*

QRS VOLTAGE CRITERIA —The QRS voltage registered by a body surface lead depends not only on the magnitude of the cardiac vector but also on the conductivity of the body tissues and the distance of the lead electrode from the heart Thus left ventricular hypertrophy occurring in a patient with emphysema which increases the distance between electrode and heart and decreases the conductivity of the lungs may fail to produce large amplitude QRS deflections Anasarca can have the same result On the other hand large QRS deflections may be recorded not infrequently in subjects with normal hearts and with thin chest walls particularly in adolescents and young adults

CRITERIA RELATING TO ORIENTATION OF PLANAR MEAN QRS VECTORS OR MEAN QRS SPATIAL VECTOR *Left-axis deviation* —Of the patients with left ventricular hypertrophy studied by Grishman and his associates only one fourth showed left axis deviation On *the other hand left axis deviation is observed in some* subjects with normal hearts and a short stocky physique In brief left ventricular hypertrophy is far from being the only factor causing left axis deviation For example Grant has presented evidence suggesting that anterolateral infarcation can be responsible for the occurrence of left axis deviation in the electrocardiogram

Leftward and posterior (clockwise) rotation of the mean horizontal QRS vector —This is commonly observed in acute and chronic cor pulmonale in which *anatomic rotation of the heart on its longitudinal axis* is believed to occur Clockwise rotation can also represent a normal variation in some individuals

CRITERIA PERTAINING TO *QRS* DURATION AND TIME OF ONSET OF INTRINSICOID DEFLECTION — Lengthening of the time required for ventricular activation can result from various types of intraventricular conduction delay in the left ventricle In fact, it can be quite difficult at times to distinguish left ventricular hypertrophy with a prolonged ventricular activation time from incomplete left bundle branch block or peri infarction block

ABNORMALLY WIDE ANGULAR DIVERGENCE OF MEAN QRS AND MEAN T SPATIAL VECTORS —Neither *the location of the leads recording depressed S–T* segments and inverted T waves nor the morphology of the S–T segments and T waves has diagnostic specificity *for left ventricular hypertrophy alone Certainly* the S–T segment deviation and T wave changes produced by digitalis coronary insufficiency hypokalemia or numerous other factors are usually indis

tinguishable from ST segment and T wave abnormalities occurring in left ventricular hypertrophy

Since none of the above criteria can individually be considered diagnostically specific for left ventricular hypertrophy it follows that the electrocardiographic diagnosis of left ventricular hypertrophy gains in reliability in direct proportion to the number of criteria satisfied

VECTORCARDIOGRAPHIC FINDINGS IN LEFT VENTRICULAR HYPERTROPHY

QRS sÊ Loop

In the patients with left ventricular hypertrophy whom we studied vectorcardographically (see Fig 91 and Table 7) the magnitude of the maximal instantaneous QRS spatial vector and the relative *size* of the spatial loop were only estimated qualitatively not measured precisely The maximal mean instantaneous *QRS vector in* left ventricular hypertrophy *tends* to be longer than that in the normal subject and the QRS sÊ loop usually encloses a larger area The configuration of the QRS sÊ loop in the frontal and horizontal projections is perhaps more variable *than in the* sagittal projection Thus the horizontal and frontal QRS loops sometimes have an oval shaped configuration in which the long and short dimensions of the loop are almost equal at other times the loops display a figure-of-eight configuration or they are extremely long and narrow

Horizontal QRS loop —In left ventricular hypertrophy the initial deflection of the horizontal QRS loop is usually written to the right and anteriorly just as occurs normally and the instantaneous vectors forming the initial deflection generally are of essentially normal magnitude However it is not uncommon to observe [illegible]
zo [illegible]
an [illegible]
fig [illegible]
te [illegible] in children and young adults with left ventricular hypertrophy than in older adults with this condition and the same seems to hold true for the corresponding electrocardiographic abnormality which consists of *relatively* deep narrow Q waves in leads I aVL, V_5 and V_6 and relatively tall initial R waves in lead V_1 (followed by the deep S waves characteristic of left ventricular hypertrophy)

Another variation in the initial deflection of the [illegible]

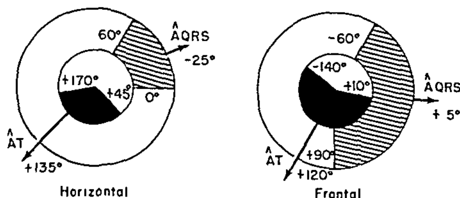
of the efferent limb almost *directly to the left and* posteriorly In other words *there* is a relative *increase* in the left right component of the initial instantaneous QRS vectors and a decrease in the anterior component of these vectors In this event the initial R waves recorded in leads V_1 and V_2 may be extremely low and may even appear to be absent but in either case leads V_5 and V_6 display normal Q waves Ordinarily when QS deflections in leads V_1 and V_2 represent residuals of an old anteroseptal myocardial infarction the normal septal Q waves are absent in leads V_5 and V_6 and this fact is *often* quite helpful in differentiating left ventricular hypertrophy and anteroseptal infarction

After inscription of the initial deflection the horizontal QRS loop in left ventricular hypertrophy is written farther posteriorly and extends farther to the left than is normally the case While this feature is present fairly constantly in most cases of left ventricular hypertrophy there is considerable variability in the configuration of the horizontal QRS loop (Fig 92) We have arbitrarily separated the various types of QRS loop configuration which we have observed in left ventricular hypertrophy into the following [illegible]

[illegible] The [illegible] ed [illegible] in a counterclockwise direction

Figure of eight left ventricular hypertrophy pattern —This type of contour is observed not infrequently in left ventricular hypertrophy The proximal loop of the "eight" is inscribed counterclock[illegible] and the [illegible] is elc[illegible] of th[illegible] [illegible] continued More often than not the initial deflection in loops showing this type of configuration is written to the left and anteriorly or less frequently posteriorly The electrocardiograms *in these* cases often show slurring of the *upstroke* of the R waves *in leads* I and V_6 and absent Q waves in these leads in addition to the findings of left ventricular hypertrophy These features would be *interpreted by some authorities as* indicating incomplete left *bundle branch block superimposed* on left ventricular hypertrophy In support of such an interpretation is the similarity of the QRS loops which we have observed and *just* described to

A Â QRS and Â T – Left Ventricular Hypertrophy



B 02 Sec and Maximal Mean Instantaneous QRS Vectors and Max T Vector – Left Ventricular Hypertrophy

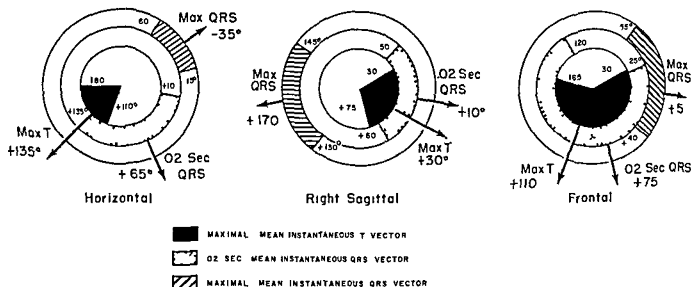


Fig 91 —A orientations of sÂ QRS and sÂ T in the horizontal and frontal planes as calculated from electrocardiograms in cases of left ventricular hypertrophy studied by the authors of this text The ranges of variation in orientation of Â QRS in the two planes are indicated by the striped segment of the outer ring of each circle of Â T in the two planes by the solid black section of the ... on of Â QRS or Â T in a given plane is indicated by ... mal mean instantaneous vector of the T ... tration) of the mean 0 02 second instantaneous vector and of the maximal mean instantaneous vector of the vectorcardiographic QRS sÊ loop in left ventricular hypertrophy The data presented in B were derived from the vectorcardiograms recorded by the authors from 35 patients with radiologic and electrocardiographic evidence of left ventricular hypertrophy

TABLE 7.—VECTORCARDIOGRAPHIC FINDINGS IN LEFT VENTRICULAR HYPERTROPHY (SEE FIG. 91 B)

QRS Class	Horizontal: Extreme Range	Horizontal: Average	Horizontal: Usual Range	Sagittal: Extreme Range	Sagittal: Average	Sagittal: Usual Range	Frontal: Extreme Range	Frontal: Average	Frontal: Usual Range
Maximal mean instantaneous QRS vector	10 to -15	35	-15 to -15	+130 to -115	+170	+155 to -170	-55 to +40	+5	-5 to +20
0.02 second mean instantaneous QRS vector	+10 to +135	+85	+20 to +100	-50 to +60	+10		-25 to -120	+75	+30 to 150
Maximal mean instantaneous T vector	+110 to 150	+135		-30 to +75	+30	-30 to +20	-30 to -105	+110	+160 to -165
Direction of initial deflection of the planar QRS loop	Right anterior			Usually anterior inferior sometimes superior			Right inferior or less often superior		
Direction of inscription of planar QRS loop	1 Counterclockwise usually 2 Less frequently in figure-of-eight loops the proximal loop of the eight is counterclockwise inscribed and the distal loop clockwise inscribed			Clockwise			Counterclockwise particularly if the initial deflection of the QRS loop is directed inferiorly		
Direction of S-T vector	Right anterior			Anterior inferior			Right inferior or occasionally superior		
Spatial QRS-T angle	85 to 175	135							
Time of appearance of maximal forward mean instantaneous QRS vector	0.01-0.05 second	0.0175 second	0.015-0.025 second						

those recorded in cases of incomplete left bundle branch block without associated left ventricular hypertrophy

Rare left ventricular hypertrophy pattern —In an occasional case of left ventricular hypertrophy a figure of eight horizontal QRS loop is observed It differs from those described above in that the initial deflection of the QRS loop forms the proximal loop of the eight while the distal portion includes the remainder of the clockwise inscribed QRS loop may be written in a clockwise or counterclockwise direction Burch and his associates found that some of the atypical frontal plane loops which were clockwise inscribed displayed a slight uptwist of their distal tips These investigators felt that this finding may be one of the earliest indications of left ventricular hypertrophy and may represent a transition stage between the normal appearing loops with clockwise inscription and the counterclockwise inscribed loops typical of left ventricular hypertrophy We have

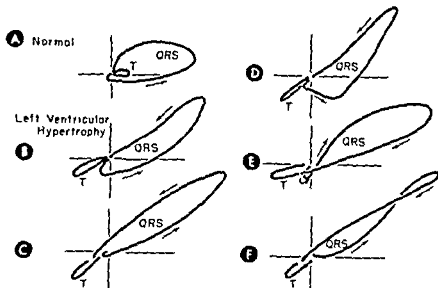


Fig 92 —*Horizontal QRS loop pattern variants in left ventricular hypertrophy* In **A** normal horizontal QRS and T loops are shown for purposes of comparison The QRS loops in **B** **C** and **D** are commonly noted in vectorcardiograms of patients with left ventricular hypertrophy The QRS loop pattern in **E** is rarely observed at least in the experience of the authors of this text The authors suspect that the QRS loop pattern in **F** may reflect combined left ventricular hypertrophy and incomplete left bundle branch block Note that in all the QRS loops shown in **B** through **F** the planar T loop is almost 180 discordant to the QRS loop

RIGHT SAGITTAL QRS LOOP —Usually the sagittal loop is initially inscribed anteriorly and inferiorly and then is written in a clockwise direction posteriorly and superiorly

FRONTAL QRS LOOP —The initial deflection of the frontal QRS loop is usually directed to the right and inferiorly and the remainder of the loop is then written in a counterclockwise direction far to the left and superiorly This produces left axis deviation of Â QRS in the electrocardiogram Sometimes the frontal and sagittal QRS loops are located inferiorly and to the left and in these cases the frontal loop may be written in a clockwise direction

Burch and his co workers using the equilateral tetrahedron system of electrode placement observed that the typical frontal QRS loop in left ventricular hypertrophy has a smooth configuration encloses a relatively wide area is inscribed in a counterclockwise direction and is located in the 0 to −60 segment of the frontal reference frame (Fig 93) The less typical loops observed in left venticular hypertrophy are located in the 0° to +60 segment and tend to have a relatively normal appearance They rarely observed transitional frontal QRS loops (recorded with the cube system) in left ventricular hypertrophy

T sÊ Loop and S–T Vector

The spatial T loop is usually directed oppositely to the QRS sÊ loop in most cases being situated to the right anteriorly and inferiorly or sometimes superiorly The angular deviation of the long axes of the *QRS sÊ and T sÊ loops ordinarily exceeds the normal* 45° ranging from 65 to 175 in the series which we studied and averaging about 155°

In left ventricular hypertrophy the QRS sÊ loop often fails to return to its point of origin after inscription an arrow drawn from the origin to the termination of the loop indicating the direction and approximate magnitude of the S–T vector The S–T vector tends to parallel the long axis of the T sÊ loop in left ventricular hypertrophy for reasons already discussed and is therefore directed to the right anteriorly and inferiorly or superiorly (Figs 94–96)

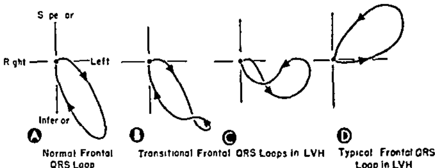


Fig 93 —Frontal QRS loop patterns recorded with the tetrahedron lead system in left ventricular hypertrophy

and his co-workers suggest that the uptwist of the tips of these loops may be an early vectorcardiographic sign of left ventricular hypertrophy which may with progression, develop into the typical pattern shown in D It should be borne in mind that these observations were made from vectorcardiograms recorded with the equilateral tetrahedron reference system rather than with the cube system

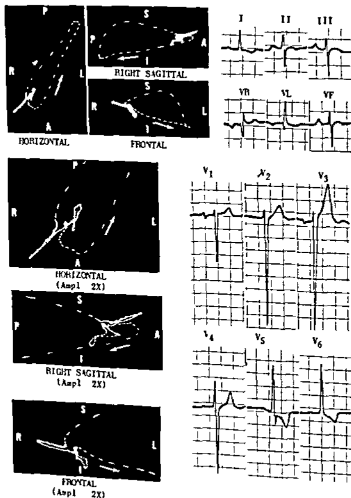
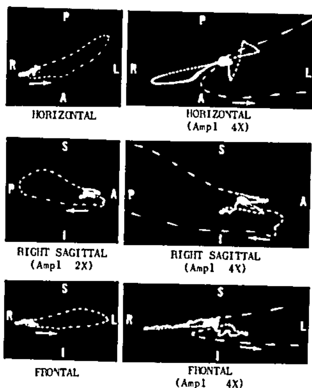
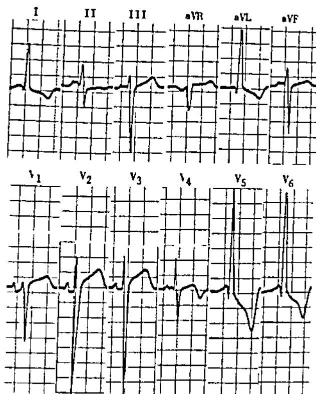


Fig 94 — Electrocardiographic and vectorcardiographic patterns of left ventricular hypertrophy

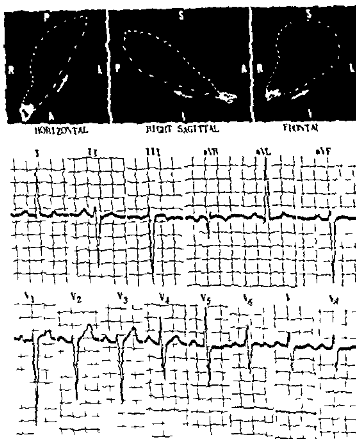
The salient diagnostic features in the electrocardiogram are (1) Left axis deviation of Â QRS = −40 (2) $S_1 + R$ = 38 mm (upper limits of normal 35 mm) (3) the spatial QRS–T angle is abnormally wide and there are inverted T waves following upright R waves in leads I V and V and upright T waves following the large downwardly directed rS deflections in the right precordial leads

The diagnostic vectorcardiographic findings are (1) Although the exact amplification of the vectorcardiogram is not stated above the smaller planar loops were recorded at a lower degree of amplification than normally indicating an increased magnitude of the mean instantaneous QRS spatial vectors (2) The posterior rotation of the long axes of the horizontal and sagittal QRS loops and the left axis deviation of the frontal loop are typical features of left ventricular hypertrophy (3) The planar QRS loops remain open after their inscription indicating an S–T vector directed to the right anteriorly and slightly superiorly (4) The long axis of the T sÊ loop is almost 160 discordant to that of the QRS sÊ loop





F 95 [illegible] hypertrophy The electrocardio voltages in leads V_5 and V_6 are the criteria for left ventricular [illegible] (R wave amplitude exceeding 11 mm with S–T depression and/or inverted T wave) and $R_I + S_{III}$ exceeds 25 mm Similarly the vectorcardiogram displays most of the features described in Figure 94 except that there is no S–T vector



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Right Ventricular Hypertrophy

Reference has already been made to the fact that the normal electrocardiogram is in essence a levocardiogram the reason being that the electrical forces *produced by the left ventricle determine* for the most part the characteristics of the QRS and T deflections in the normal electrocardiogram Left ventricular hypertrophy simply exaggerates the normal electrical preponderance of left over right ventricle so that the general form of the QRS deflections in the various leads is not strikingly altered although ordinarily *the size of the QRS complexes is greatly increased* (The shortcomings of QRS voltage criteria for the diagnosis of the left ventricular hypertrophy were considered in Chapter 9)

Right ventricular hypertrophy on the other hand tends to cause a marked change in the balance of cardiac forces and therefore typically at least is *accompanied by characteristic alterations in the appear*ance of the QRS deflections Thus by and large the typical features of right ventricular hypertrophy are more readily recognized and are more reliable diagnostic signs than those of left ventricular hypertrophy However for right ventricular hypertrophy to produce recognizable changes in the electrocardiogram and vectorcardiogram it is not enough that the right ventricular potentials merely increase in magnitude The increment in the magnitude of the right ventricular forces must be such that the normal electrical predominance of the left ventricle is abolished or at *least reduced to the degree that specific changes in* QRS configuration or voltage appear in the electrocardiogram This being the case it is not surprising that right ventricular overloading must exist for a considerable period of time if the pattern of right ventricular hypertrophy is to develop in the electrocardiogram and vectorcardiogram even then often enough diagnostic changes may fail to appear

The prominence of the electrocardiographic and vectorcardiographic manifestations of right ventricular hypertrophy when present depends on among other things how early in the QRS interval and to *what degree right ventricular forces offset and even*tually dominate forces arising in the left ventricle While these factors are related to a great extent to the type of cardiac lesion producing the right ventricular hypertrophy for the present the discussion will be limited to the general patterns of right ventricular hypertrophy without regard to their specific etiology

THE INSTANTANEOUS VA VECTORS

The instant to instant changes in the balance of *cardiac forces in right ventricular hypertrophy* and the manifestations of these changes in the electrocardiogram can be illustrated in a simplified fashion by utilizing the hypothetical instantaneous VA vectors to schematize the ventricular activation process as was done in the preceding chapter on *left ventricular hypertrophy* To relate better this general discussion of typical electrocardiographic findings to subsequent more specific descriptions of the electrocardiogram and vectorcardiogram in right ventricular *hypertrophy two patterns or series of* VA vectors will be considered each having its electrocardiographic and vectorcardiographic counterpart as will be seen later We have arbitrarily designated these two patterns as the *RSR* and the *tall R* patterns of right ven*tricular hypertrophy with the purpose of suggesting* by these terms the characteristics of the instantaneous VA or QRS vectors which produce RSR deflections in electrocardiographic lead V_1 in some cases of right

ventricular hypertrophy and tall R waves in that same lead in other cases For the same reason these terms will be applied to the corresponding vectorcardiographic patterns of right ventricular hypertrophy

The Tall R and the RSR Patterns of Right Ventricular Hypertrophy

TALL R PATTERN OF RIGHT VENTRICULAR HYPERTROPHY	RSR PATTERN OF RIGHT VENTRICULAR HYPERTROPHY
Th ... for example	In this pattern of right ventricular hypertrophy (Fig 98) the instantaneous mean vectors appearing during the first 0 04 second of the QRS interval are essentially normal in direction and magnitude and project an rS deflection on lead V Subsequent instantaneous vectors develop in a rightward and anterior direction but do not attain a magnitude comparable to that of the earlier leftward vectors even though they produce a prominent terminal R wave in lead V Lead V therefore registers an rSR deflection The RSR pattern of right ventricular hypertrophy tends to be observed electrocardiographically and vectorcardiographically when anatomically there is selective hypertrophy of basal portions of the right ventricle and of the trabecular network as opposed to concentric hypertrophy of the entire right ventricular wall The dilatation of the right ventricle which usually accompanies selective hypertrophy may be an additional factor in the production of the RSR pattern of right ventricular hypertrophy

0 01 Second Septal VA Vector

1 Initially the interventricular septum is activated just as normally so that the septal VA vector is directed to the right anteriorly and inferiorly or superiorly

Leads I and V	Small normal Q wave
Lead aVF	Small normal Q wave if the septal vector is directed *superiorly* or the beginning upstroke of an R wave if the vector is directed inferiorly
Lead V	Beginning upstroke of an initial R wave

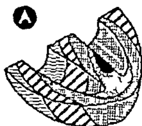
2 Occasionally in the tall R ... lar the ort of

Leads I and V	Beginning upstroke of an initial R wave
Lead aVF	(As in paragraph 1 above)
Lead V	Small Q wave

0 02 Second VA Vector

TALL R PATTERN	RSR PATTERN
At ... th of ma inf the ... the tall R pattern of right ventricular hypertrophy	During activation of the apicoanterior and apicodiaphragmatic walls of the left and right ventricles the left ventricular forces are electrically preponderant to much the same degree as normally *Thus the 0 02 second VA vector is directed inferiorly anteriorly and to the left*

(Continued on p 142)



Sequence of Septal-Ventricular Activation in Right Ventricular Hypertrophy

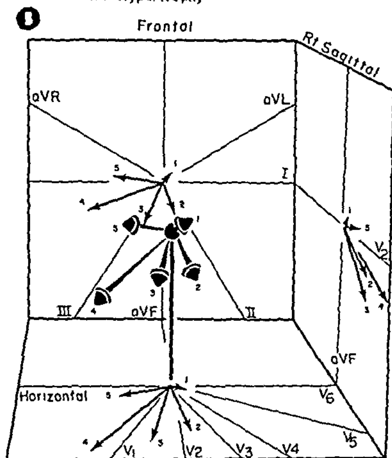
1 INITIAL ACTION OF LEFT SEPTAL SURFACE

2 CONTINUED SEPTAL ACTIVATION AND ACTIVATION OF APICO ANTERIOR FREE WALL OF BOTH VENTRICLES

3 ACTIVATION OF ANTEROLATERAL FREE WALL OF BOTH VENTRICLES

4 CONTINUED ACTIVATION OF ANTEROLATERAL WALL OF RIGHT VENTRICLE AND SEPTUM AND ACTIVATION OF BASAL WALL OF LEFT VENTRICLE

5 COMPLETION OF ACTIVATION OF BASAL SEPTUM AND RIGHT VENTRICLE FREE WALL

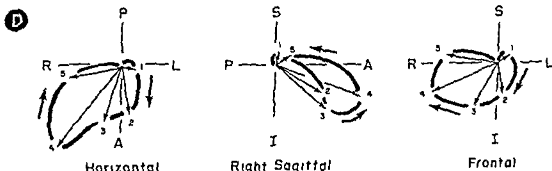


Instantaneous VA Vectors in Right Ventricular Hypertrophy — Tall R Pattern



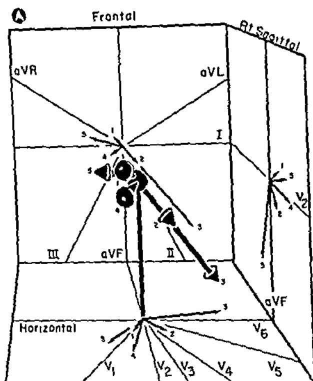
V_1 V_2 V_3 V_4 V_5 V_6

QRS Deflections Projected on Scalar Leads

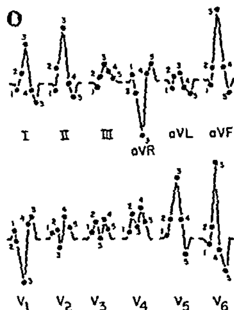


Planar QRS Loops in Right Ventricular Hypertrophy

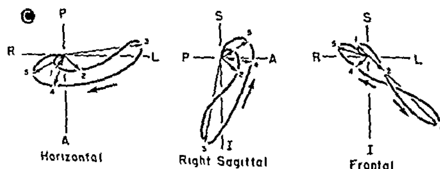
Fig 97 —Legend on facing page



Instantaneous VA Vectors in Right Ventricular Hypertrophy - RSR Pattern



QRS Deflections Projected on Scalar Leads



Planar QRS Loops in Right Ventricular Hypertrophy

Fig 98 —Instantaneous VA vectors in the RSR –

F g 97

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loops ... scription of the sagittal loop and the anterior and rightward orientation of th planar ... scription of the horizontal QRS



Sequence of Septal-Ventricular Activation in Right Ventricular Hypertrophy

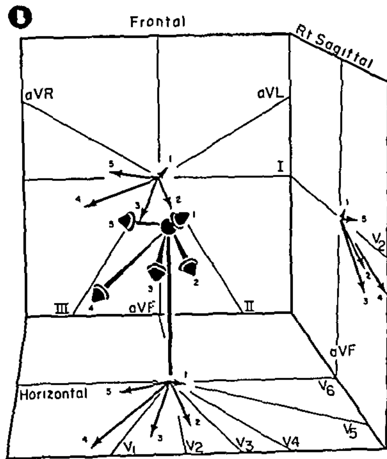
1 INITIAL ACTION OF LEFT SEPTAL SURFACE

2 CONTINUED SEPTAL ACTIVATION AND ACTIVATION OF APICO ANTERIOR FREE WALL OF BOTH VENTRICLES

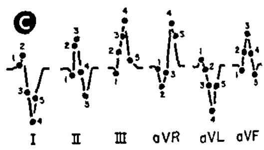
3 ACTIVATION OF ANTEROLATERAL FREE WALL OF BOTH VENTRICLES

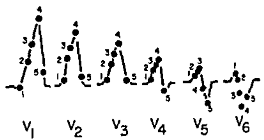
4 CONTINUED ACTIVATION OF ANTEROLATERAL WALL OF RIGHT VENTRICLE AND SEPTUM AND ACTIVATION OF BASAL WALL OF LEFT VENTRICLE

5 COMPLETION OF ACTIVATION OF BASAL SEPTUM AND RIGHT VENTRICLE FREE WALL

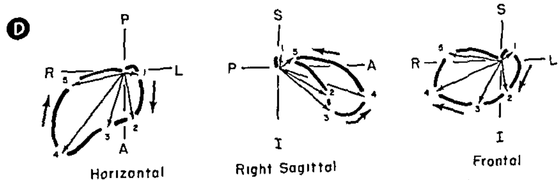


Instantaneous VA Vectors in Right Ventricular Hypertrophy — Tall R Pattern





QRS Deflections Projected on Scalar Leads



Planar QRS Loops in Right Ventricular Hypertrophy

Fig 97 —Legend on facing page

SUMMARY OF QRS CONFIGURATIONS

Leads I and V_6	qrS rS	Leads I and V_6	qRs
Lead aVF	qrS qRs RS Rs	Lead aVF	qRs qR Rs R
Lead V_1	rsR rR qR	Lead V_1	rSR rSr'

QRS Configuration in the Precordial Leads in the Tall R and RSR Patterns of Right Ventricular Hypertrophy

The orientation of the 0 01 second septal VA vector and 0 02 second apicoanterior VA vector with respect to the lead axis of V_1 determines whether the tall R wave in this lead is preceded by a small initial R wave (rR complex) by a small Q wave (qR complex) or by embryonic R and S waves (rsR) or merely displays slurring or notching of its upstroke (Fig 99) Sometimes the tall R wave in lead V_1 is f llo ed b

lead V_1 such as those cited above are discussed in greater detail in the section dealing with the vector cardiographic tall R pattern.

While the specific mechanism responsible in right ventricular hypertrophy for the *rotation of the instantaneous VA vectors to the right and anteriorly or less commonly posteriorly* is not known the effects of this rotation are readily apparent

1 As indicated previously the fact that the VA

Normally right ventricular activation is dominated almost completely by the depolarization forces of the left ventricle so that with the exception of the 0 01 and 0 02-second VA vectors virtually all of the VA vectors tend to be directed to the left and poste

ized by a del
vector for le
dom nance o ... ght ventricle does not usually make its appearance until later in the QRS interval Accordingly onset of the intrinsicoid deflection in lead V_1 is also late (0 04 second or more)

3 As will be seen later the displacement of the VA vectors increasingly to the right anteriorly and in a superior direction produces the two vectorcardiographic findings most consistently present in right ventricular hypertrophy—namely clockwise inscription of the QRS sE loop in the horizontal projection and counterclockwise inscription of the spatial loop in the sagittal projection

As previously indicated qR complexes may occasionally be recorded in lead V_1 in right ventricular hypertrophy Sometimes the Q wave in this lead can be attributed to the fact that the initial 0 01 second VA vector is oriented relatively perpendicular to the lead axis of V_1 Thus the initial R wave of what would

leftward initial deflection of the QRS sE loop in the vectorcardiogram Some of the mechanisms which have been proposed in explanation of this latter finding are as follows

1 The leftward deflection of the QRS sE loop has been interpreted as signifying *initial* excitation of the right septal surface and subsequent septal activation in a direction the reverse of normal The proponents of this theory believe that right to-left septal depolarization also occurs in a small percentage of normal subjects

2 On the other hand the leftward orientation of the initial QRS vectors has been attributed by other authorities to increasing degrees of clockwise rotation of a vertical heart around its longitudinal axis

3 It has also been postulated that the initial negativity of the right septal surface may be due to a decrease in certain areas of the *density* of the union between Purkinje fibers and muscle fibers as the result of dilatation of the ventricle

4 Still another theory proposes that potentials arising in the high basal portion of the septum are transmitted to the right precordium through a dilated right atrium This region of the septum is activated late in systole and dominated earlier in the QRS interval by potentials produced by activation of free ventricular wall the *former* factor accounting for the late R wave and the latter for the Q wave in lead V_1

In the tall R pattern of right ventricular hypertrophy the chest leads show a reversal of the normal precordial QRS transition that is to say instead of the normal progressive increase in the R/S amplitude ratio from right to left across the precordium the

Leads I and V_6	Beginning upstroke of an R wave
Lead aVF	Upstroke of an R wave
Lead V_1	Isoelectric segment or a minor upward or downward deflection depending on minor differences in the position of the 0.02 second VA vector

Leads I and V_6	Beginning upstroke of an R wave
Lead aVF	Upstroke of an R wave or beginning upstroke of an R wave following a small Q wave
Lead V_1	Descending limb of a small initial R wave or the beginning downstroke of the following S wave

0.04 Second VA Vector

Shortly before the appearance of the 0.04 second VA vector and much earlier in the period of ventricular activation than is the rule in the RSR pattern of right ventricular hypertrophy the hypertrophied right ventricle in the tall R pattern of right ventricular hypertrophy begins to assert its electrical predominance as evidenced by the development of subsequent instantaneous vectors in a superior anterior and rightward direction Thus the 0.04 second VA vector in this right ventricular hypertrophy pattern extends only slightly inferiorly and to the left or extends slightly to the right In either case it projects far anteriorly

Leads I and V_6	Beginning downstroke of a deep S wave if the vector lies to the right or the descending limb of an R wave if it lies to the left
Lead aVF	Descending limb of the R wave
Lead V_1	Beginning upstroke of a tall R wave

Although the electrical predominance of the left ventricle continues to be maintained it is not so complete as is normally the case since the opposing right ventricular forces of increasing magnitude counterbalance a larger and larger fraction of the left ventricular forces in contrast with the normal course of events In consequence the resultant of the component left and right ventricular forces the 0.04 second VA vector is of lesser magnitude than normal and extends less to the left and posteriorly or may even lie slightly anteriorly Generally however this vector remains the maximal mean instantaneous VA vector just as it is normally

Leads I and V	Peak of the R wave
Lead aVF	Completion of the upstroke or descending limb of R wave
Lead V_1	Nadir of S wave (The S wave in V_1 may be shallower than the normal S wave in this lead because of the more anterior orientation of the 0.04 second VA vector in the RSR pattern of right ventricular hypertrophy)

0.06 Second VA Vector

As the relative strength of the left ventricular potentials continues to wane the preponderance of right ventricular forces becomes increasingly marked As a result the 0.06 and 0.08 second VA vectors are of increased magnitude and come to be oriented more and more toward the effective electrical site of the right ventricle—i e superiorly to the right and anteriorly

Leads I and V	Terminal deep S wave of greater size than the preceding R wave
Lead aVF	Terminal S wave which may or may not be larger than the preceding R wave
Lead V	Completion of tall terminal R wave which is the sole component wave or the dominant component of the ventricular deflection in lead V

During the terminal half of the ventricular activation period right ventricular forces begin to exceed left ventricular forces and as this progresses the instantaneous VA vectors rotate anteriorly superiorly or upward and to the right Thus the 0.06- and 0.08 second VA vectors assume an anterior inferior and slightly leftward or increasingly rightward position

Leads I and V	Terminal S wave which is smaller than the preceding R wave
Lead aVF	Completion of R wave or small terminal S wave
Lead V_1	Completion of terminal R wave which may or may not be larger than the preceding S wave

R/S ratio is greater in V_1 than in V_6 and decreases from right to left across the precordium (Fig 100) The reversed precordial transition in right ventricular hypertrophy results from the displacement of the instantaneous vectors toward the right and anteriorly away from their normal location to the left and posteriorly This abnormality becomes increasingly obvious in the precordial electrocardiogram as the orientation of the maximal VA vector (or Â QRS in the horizontal plane) approaches the positive axis of lead V (+90) and quite marked when the maximal vector shifts to the right of this point

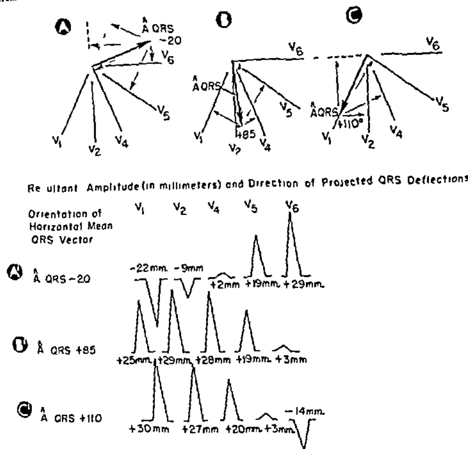
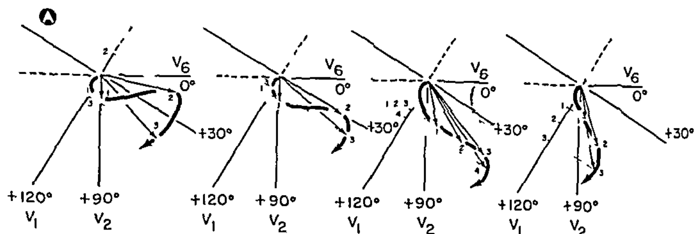
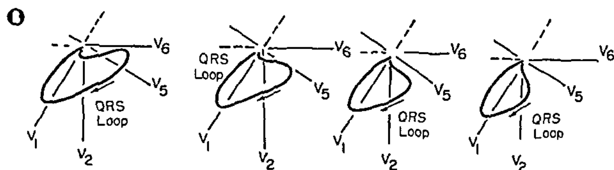


Fig 100 –Explanation of reversed precordial QRS transition in right ventricular hypertrophy It should be remembered that the projection of a mean vector on a lead axis yields information only in terms of the net polarity and net area or voltage of QRS In A the mean QRS vector occupies a normal position in the horizontal plane at about −20 and as can be seen in A projects a normal precordial QRS transition on the chest leads From V_1 to V there is a steadily

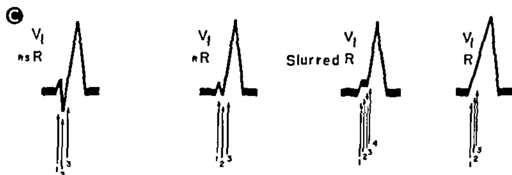
… resultant positivity on right precordial leads (C) and resultant negativity on left precordial leads the result being a striking reversal in the precordial QRS transition The arbitrarily selected length of the mean QRS vector is the same in A B and C The measured projection of the mean QRS vector on each lead axis is expressed in millimeters From the latter values were derived the resultant amplitudes of the schematic deflections depicted in A B and C



Variations in course of the early efferent limb of the horizontal QRS loop in RVH



Corresponding configurations of the horizontal QRS loops in RVH



Related variations in the initial portion of the QRS deflection in Lead V_1

Fig 99 —The effect of variations in the course of the efferent limb of the horizontal QRS loop in right ventricular hypertrophy on the QRS configuration in lead V_1 ... QRS planar vectors and their projections on the lead ... from which the ... projections of ... wave recorded in lead V_1 Note that in the fir ... the third there is a slur on the upstroke of the R wave and in the fo ... these striking changes in QRS configuration in lead V_1 ... characteristics of vectors *1–3* and corresponding parts of the QRS loop

Orientation of Â QRS

Frontal Plane

The orientation of Â QRS in the frontal plane was calculated by the authors of this text from electrocardiograms of 90 patients with vectorcardiographic right ventricular hypertrophy due to congenital heart disease mitral stenosis or chronic cor pulmonale

	Range	Average
Congenital heart disease	+75 to −150	+130
Mitral stenosis	0° to +140	+100
Chronic cor pulmonale	+120 to −90	+145

Horizontal Plane

The orientation of Â QRS in the horizontal plane was not calculated by the authors because it was not possible to do so with any degree of accuracy in many instances

Lengthened activation time of the hypertrophied right ventricle

1 On ... he QRS interval (normal)

Rotation of the mean instantaneous T spatial vectors away from the mean instantaneous QRS spatial vectors

EXTREMITY LEADS

Leads I and aVL record upright T waves and leads II III aVR and aVF usually register inverted T waves and depressed ST segments

PRECORDIAL LEADS

Lead V usually displays inverted T waves and depressed ST segments while the T waves are upright in lead V_4

Related T Wave Criteria

Leads V_1 and V record inverted T waves with R waves of 5 mm or more amplitude

QRS criteria for the diagnosis of right ventricular hypertrophy recommended by Milnor

1 R/S or R/S ratio in V_1 greater than 1 with R or R greater than 0 5 mv and a QRS duration less than 0 12 second

2 Milnor considers right axis deviation of Â QRS to be diagnostic of right ventricular hypertrophy if Â QRS is situated to the right and between +110 and −91

The ...

QRS criteria for the diagnosis of right ventricular hypertrophy recommended by the authors of this text

1 An R S R/S ...

3 Right axis deviation of Â QRS to the right of +110

4 An R/S ...

VENTRICULAR REPOLARIZATION

For essentially the same reasons as applied in the case of left ventricular hypertrophy secondary and *less commonly* primary T wave abnormalities occur in right ventricular hypertrophy When the right ventricular myocardium is hypertrophied a longer time is required for activation to spread from endocardium to epicardium Consequently endocardial layers of muscle are able to recover earlier than subepicardial muscle with the result that repolarization spreads *through the right ventricular wall* in a direction the reverse of normal Repolarization forces arising in the right ventricle therefore summate with those produced in the left ventricle and cause the instantaneous T vectors almost always to be directed away from the terminal instantaneous VA (QRS) vectors and less frequently away from the maximal instantaneous VA (QRS) vector Thus the mean or maximal T vector is usually directed to the left posteriorly and superiorly in the RSR pattern and less *to the left and* more posteriorly and inferiorly in the tall R pattern

In short when there is an upright terminal component of the QRS deflection in V_1 the *following* T wave is generally inverted when there is a terminal S wave in V_1 the T wave may be upright Prominent S–T segment depression in right precordial leads is not so common in right ventricular hypertrophy as is S–T segment depression in the left precordial leads in left ventricular hypertrophy but when present in the former it is usually associated with the tall R pattern of right ventricular hypertrophy

GENERAL ECG FINDINGS AND RELATED DIAGNOSTIC CRITERIA IN RIGHT VENTRICULAR HYPERTROPHY

Increased magnitude and/or more anterior superior and rightward orientation of the mean instantaneous QRS spatial vectors

EXTREMITY LEADS

1 Leads I and aVL record RS or rS deflections while leads II III and aVF commonly record qR qRs or Rs deflections or less commonly in marked right axis deviation these leads display rS complexes There is usually a tall terminal R wave in lead aVR

PRECORDIAL LEADS

1 Although the configuration of the QRS complex recorded in lead V_1 may vary widely from case to case in right ventricular hypertrophy nevertheless in virtually every diagnosable case the resultant voltage of the deflection has a positive sign In other words whether the QRS deflection in lead V has an rsR qR Rs or rR configuration in each instance the QRS deflection is predominantly upright Lead V_6 may record a QRS deflection of essentially normal size and configuration in right ventricular hypertrophy but when the electrocardiographic pattern of right ventricular hypertrophy is well developed this lead generally registers an equiphasic RS deflection or an rS or qrS deflection

Related QRS Voltage Criteria

R in aVR $\geqq$ 5 mm

R in V $>$ 7 mm
S in $V_1 \leqq$ 2 mm
R/S ratio in V $>$ 1
R in V_1 + S in V_5 or V $>$ 10 5 mm
R in V or V $<$ 5 mm
S in V or $V_6 \geqq$ 7 mm
R/S ratio in V or V $\leqq$ 1

2 There is usually right axis deviation of the mean manifest electrical axis of QRS Thus lead I usually registers an essentially downward ventricular deflection If leads II and III record upright QRS deflections then the electrical axis or mean frontal QRS vector is situated in

frame
QRS de
an QRS

vector lies to the right and above the −150 axis of the frontal reference frame In such an instance the bipolar limb leads show the S_I–S_{II}–S_{III} pattern each lead recording a QRS deflection whose largest component is an S wave

2 Since the mean instantaneous QRS spatial vectors are displaced to the right and anteriorly in right ventricular hypertrophy the corresponding mean horizontal QRS vector is rotated accordingly *and tends to assume a position* almost 180 away from its normal location Consequently in right ventricular hypertrophy there is a reversed precordial QRS transition as evidenced by a right to left progressive decrease in the R/S amplitude ratio

tion of the loop a relatively minor limb is then written to the left and anteriorly its limited leftward extent reflecting earlier onset of right ventricular preponderance in comparison with the RSR pattern of right ventricular hypertrophy If the leftward limb of the loop extends into the 0 to +30 segment of the horizontal reference frame lead V_1 records a small S wave if it reaches the +30 axis without passing beyond the initial R wave in V_1 is followed by a notch which does not descend below the isoelectric base line (Fig 99) if the leftward deflection is written almost perpendicular to the lead axis of V_1 a slur appears on the upstroke of the tall R wave subsequently inscribed in this lead and finally if the deflection continues to be written increasingly anteriorly as well as momentarily to the left lead V_1 registers the beginning of the upstroke of a tall R wave After inscription of the short leftward segment the loop abruptly swings more anteriorly and to the right and returns in a clockwise direction to

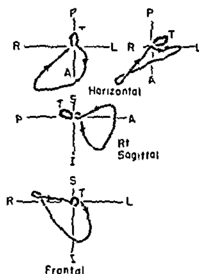
TABLE 8 —QRS sÊ AND T sÊ LOOP FINDINGS IN THE TALL R AND RSR PATTERNS OF RIGHT VENTRICULAR HYPERTROPHY

Projection	Initial Deflection of QRS sÊ Loop	Maximum Mean Instantaneous QRS Vector		Direction of Inscription of QRS sÊ Loop	Configuration of QRS sÊ Loop	Terminal Mean Instantaneous Vector of QRS sÊ Loop	Maximum Mean Instantaneous T Vector
		Range	Axis				
Horizontal	Right, anterior or left anterior	Tall R +15 to +170	+1[illegible]0	Clockwise	Triangular or ovoid	Right anterior or posterior	Left posterior
		RSR −10 to +10	+5	1 Clockwise 2. Counter clockwise clockwise	1 J shaped efferent and afferent limbs 2 Figure-of eight loop configuration	Anterior right	Left, posterior
Right sagittal	Anterior superior or anterior inferior	Tall R −7[illegible] to +80	+40	1 Clockwise-counter clockwise 2. Counter clockwise	1 Figure-of eight with small proximal loop 2 Elongated or oval	Anterior inferior or anterior superior	Posterior inferior or posterior superior
		RSR +70 to +115	+80	Clockwise-counter clockwise	Figure-of-eight	Anterior inferior	Posterior inferior or superior
Frontal	Right, inferior or right, superior Occasionally left inferior or left, superior	Tall R +20 to 130	+130	Clockwise	Variable	Right inferior or right superior	Left inferior or left superior
		RSR +10 to +90	+60	Counter clockwise or clockwise	Variable		

VECTORCARDIOGRAPHIC FINDINGS IN RIGHT VENTRICULAR HYPERTROPHY

Before considering the QRS sE loop configurations which correspond to the two arbitrary patterns of right ventricular hypertrophy sketched earlier in this chapter in terms of the instantaneous VA vectors and electrocardiogram, it is well to preview the vectorcardiographic abnormalities most typically and consistently observed in right ventricular hypertrophy (see also Fig 101 and Table 8). By and large all of the variant QRS sE loop patterns produced by right ventricular hypertrophy share in common the following general abnormalities:

1. The QRS sE loop is situated more anteriorly than is normally the case.
2. The long axis of the QRS sE loop tends to be rotated medially or to the right.



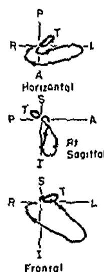


Fig 101 —Schematic planar QRS and T loops depicting the vectorcardiographic characteristics of the RSR and tall R patterns of right ventricular hypertrophy

3. The farther to the right the rotation of the QRS sE loop, the less inferior or the more superior is its location, as a general rule.
4. The afferent limb of the QRS sE loop, either in whole or in part, is written in a rightward, anterior, and superior direction, and this causes corresponding portions of the horizontal and right sagittal QRS loops to be inscribed in clockwise and counterclockwise directions, respectively.

Vectorcardiographic Tall R Pattern

The essential characteristics of this QRS sE loop pattern of right ventricular hypertrophy (see also Figs 101 and 102) can be listed as follows:

1. The long axis or maximal mean instantaneous vector of the QRS sE loop is oriented either far to the right and somewhat anteriorly (occasionally posteriorly) or almost directly anteriorly and slightly to the right.
2. Alternatively, the mean instantaneous vectors of the QRS sE loop may extend anteriorly and equally to the left and right of the midline.
3. The mean instantaneous vectors of the QRS sE loop which lie to the right and anteriorly are of much greater magnitude than corresponding vectors in the RSR loop pattern of right ventricular hypertrophy, which will be described later. These large rightward and anterior vectors are responsible for the tall R wave recorded in lead V_1 of the electrocardiogram.

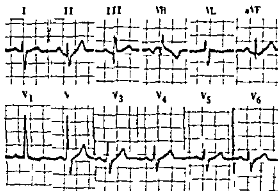
The planar QRS loops of the vectorcardiogram in ... are

... por-
jection the QRS sE loop can have an initial deflection directed to the right and anteriorly or one directed to the left and slightly anteriorly or posteriorly. (Some of the theories which have been proposed to account for the apparent reversed direction of the septal activation forces were considered earlier.) Following the initial deflec-

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septal activation produces forces directed to the left and posteriorly (a not infrequent f d ... h ...



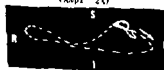
teriorly as is the maximal mean instantane

... (3) he frontal QRS loop is rotated inferiorly and medially and lies at about +75









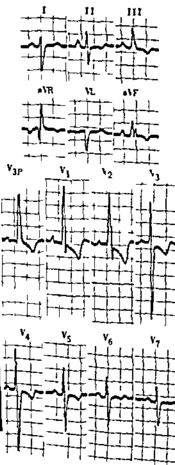


Fig 104 —Electrocardiographic and vectorcardiographic tall R pattern of right ventricular hypertrophy recorded in a man 30 with cyanotic congenital heart disease of unknown type

Electrocardiographic findings supporting the diagnosis of right ventricular hypertrophy are (1) There is right axis deviation of $\hat{A}$ QRS (+150) (2) The terminal R of the QR deflection in aVR is greater than 5 mm amplitude (3) There are small Q and tall R waves (18 mm) with late onset of intrinsicoid deflection (0 07" second) in lead V₁ and a reversed precordial QRS transition (4) The T waves are inverted in the precordial leads showing upright QRS deflections which signifies an abnormally wide QRS–T spatial angle

The diagnostic vectorcardiographic abnormalities are (1) After the initial inscription of the QRS sE loop to the left slightly posteriorly and inferiorly the loop is written primarily anteriorly and to the right in a clockwise direction in the horizontal projection and in a counterclockwise direction in the sagittal projection The maximal mean instantaneous QRS spatial vector is situated to the right anteriorly and inferiorly (2) There is a small S–T vector directed to the right (3) The T sE loop is almost 180 discordant to the QRS sE loop (4) The anterior orientation of the P sE loop is suggestive of right atrial enlargement

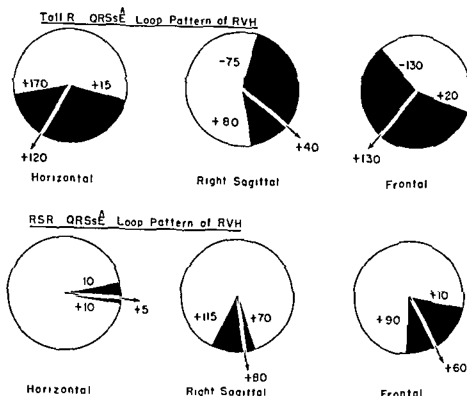


Fig 102 —Extreme range of variation and average orientations of the maximal mean instantaneous QRS planar vectors of the QRS sÊ loops in the tall R and the RSR vectorcardiographic patterns of right ventricular hypertrophy

its point of origin The maximal instantaneous QRS vector tends to lie in the right anterior quadrant Since most of the larger instantaneous QRS vectors are situated anterior and in the case of the later vectors to the right tall R waves are projected on leads V_{3R} V_1 and V and S waves on V_6

b) Right sagittal QRS loop —Following an initial deflection anteriorly and slightly superiorly the QRS sÊ loop in the sagittal projection is usually written in a counterclockwise direction anteriorly and at first inferiorly and later slightly superiorly Occasionally the greater part of the sagittal QRS loop is situated superiorly

c) Frontal QRS loop —After inscription of the initial deflection which can be directed to the right or left (see discussion of horizontal QRS loop above) the frontal QRS loop is usually written in a clockwise direction to the left and inferiorly and then returns on the right and inferiorly or somewhat superiorly Since the loop projects farther to the right than to the left in this pattern lead I registers an rS deflection and leads II III and aVF R waves or RS deflections Occasionally the QRS sÊ loop in the frontal projection is inscribed in a counterclockwise direction to the left and superiorly and then to the right In this instance all limb leads register rS deflections except lead aVR which records a QR complex

Vectorcardiographic RSR Pattern

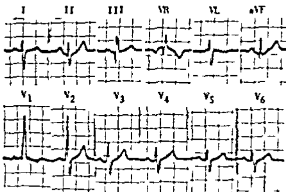
The theories as to the manner in which this vectorcardiographic pattern of right ventricular hypertrophy (and the corresponding electrocardiographic pattern) is produced are as varied as they are numerous Some authorities attribute the terminal R to delayed spread of activation through a right ventricular wall of normal thickness but with greatly hypertrophied trabeculae Others consider the late rightward forces to be due to incomplete right bundle branch block possibly secondary to right ventricular dilatation Perhaps the most attractive theory and certainly the theory favored by the weight of evidence is that the late rightward and anteriorly directed forces in the RSR pattern of right ventricular hypertrophy are produced by activation of hypertrophied basal regions of the right ventricle including the crista supraventricularis its parietal and septal bands and the trabecular network of the right ventricle Selective hypertrophy of these regions unac

F 103 El ctroc d og ph n l ne



u ... ett and posteriorly (a not infrequent finding in right ventricular hypertrophy)

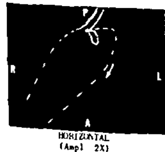
The diagnostic vectorcardiographic findings are (1) The QRS sE loop is displaced ...



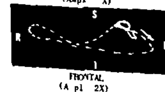
teriorly as is the m m l m n n ...

...) (3) The frontal QRS loop is rotated inferiorly and medially and lies at about + o









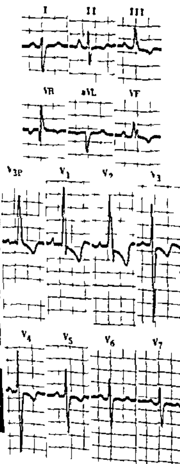


Fig 104 –Electrocardiographic and vectorcardiographic tall R pattern of right ventricular hypertrophy recorded in a man 30 with cyanotic congenital heart disease of unknown type

Electrocardiographic findings supporting the diagnosis of right ventricular hypertrophy are (1) There is right axis deviation of Â QRS (+150) (2) The terminal R of the QR deflection in aVR is greater than 5-mm amplitude (3) There are small Q and tall R waves (18 mm) with late onset of intrinsicoid deflection (0 07 second) in lead V1 and a reversed precordial QRS transition (4) The T waves are inverted in the precordial lead showing upright QRS deflections which signifies an abnormally wid QRS–T spatial angle

The diagnostic vectorcardiographic abnormalities are (1) After the initial inscription of the QRS sE loop to the left slightly posteriorly and inferiorly the loop is written primarily anteriorly and to the right in a clockwise direction in the horizontal projection and in a counterclockwise direction in the sagittal projection The maximal mean instantaneous QRS spatial vector is situated to the right anteriorly and inferiorly (2) There is a small S–T vector directed to the right (3) The T sE loop is almost 180 discordant to the QRS sE loop (4) The anterior orientation of the P sE loop is suggestive of right atrial enlargement

HORIZONTAL CUBE

HORIZONTAL TETRAHEDRON

RIGHT SAGITTAL CUBE

RIGHT SAGITTAL TETRAHEDRON

FRONTAL CUBE

FRONTAL TETRAHEDRON

I II III aVR aVL aVF V_1 V_2 V_3 V_4 V_5 V_6

Fig 105 — Electrocardiographic and vectorcardiographic RSR pattern of right ventricular hypertrophy in a man 28 ... alvular lesions

(1) The P waves in the bipolar limb leads are notched or slurred

... upported by the ...

the diphasic P wave in lead V_1 Since the ... wave transition occurs at about lead V_4 Â P is directed approximately along the −15 axis of the horizontal reference fr... ...ent with the diagnosis of P mitrale (2) Minimal right axis deviatio... ...ll rsR complex of 0 09 second duration and in R amplitude of 4 ... abnormal although V... shows a relatively deep S wave (4) The T ... entricular hypertrophy

been interpreted in the past as being indicative of incomplete (described below) discloses the error in this diagnosis The vec... was recorded with the cube system of electrode placement (just ...) and the second vectorcardiogram was obtained with the equ...

...here is an S-T vector directed to the right posteriorly ... l vector of the QRS sÊ loop is directed to the left in ... terminal QRS vectors (1) The P sÊ loop is extremely large in each projection and is oriented ... pos... , ... , and only slightly to the left The configuration of the P sÊ loop is characteristic of P mitrale the horizontal loop is a figure-of eight the larger component of the loop being

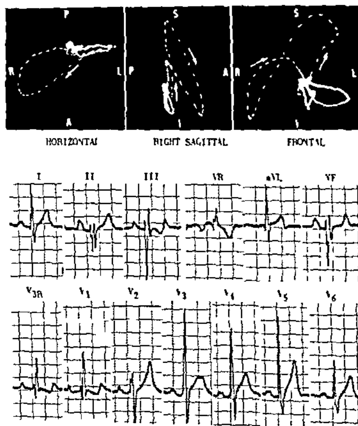


Fig 106 — Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy in a boy 15 with surgically proved interatrial septal defect

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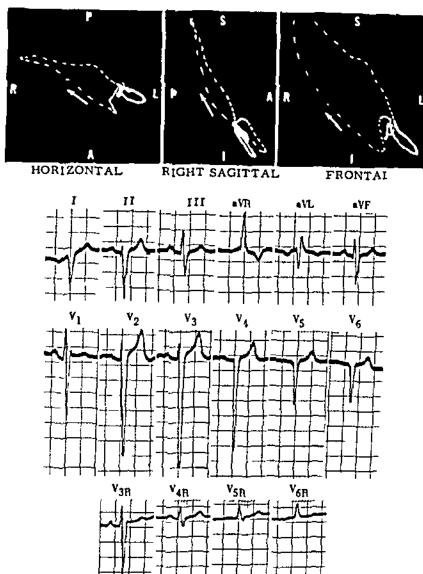


Fig 107 —Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy in a youth 21 with undiagnosed cyanotic congenital heart disease The electrocardiogram displays the S_I–S_{II}–S_{III} pattern which is most commonly observed in chronic cor pulmonale

The electrocardiographic findings of significance are these (1) Â QRS = −150 in the frontal plane Â T = +60 *Â P = +120* (2) *All limb leads display resultantly negative QRS deflections except for lead III which shows* an equiphasic RS deflection and lead aVR which records a tall R wave (3) Â QRS = −135 in the horizontal plane There is a reversed precordial QRS transition with resultant positivity on the right (leads V R to V R) and resultant negativity on the left

– orly and posteriorly

for ... RS loop is clockwise

insc

companied by more generalized hypertrophy of the right ventricular wall has been observed pathologically in association with cardiac lesions which increase right ventricular stroke volume

The essential characteristics of *the RSR type of* QRS sÊ loop configuration (Figs 101 and 102) are as follows

1 The long axis or maximal mean instantaneous vector of the QRS sÊ loop is oriented to the left and slightly anteriorly or posteriorly and has much the same magnitude as normally
2 The efferent limb of the QRS sÊ loop is inscribed more or less normally but the entire afferent limb or the terminal half of the afferent limb is written to the right and anteriorly with a reversed direction of inscription in the horizontal and sagittal projections

The more detailed features of the planar QRS loops in the RSR pattern of right ventricular hypertrophy are presented in the following paragraphs

a) Horizontal QRS loop —In the horizontal projection the QRS sÊ loop moves at first to the right and anteriorly the initial deflection resembling that of the normal QRS sÊ loop The loop is then written to the left posteriorly or slightly anteriorly and inferiorly In general the onset of right ventricular electrical predominance is signaled by a deflection of the QRS sÊ loop in an anterior rightward and superior direction When the horizontal QRS loop reaches its turning point or maximal leftward extent instead of turning in a counterclockwise direction posteriorly the loop rotates anteriorly and is then written in a clockwise direction from left to right. Thus the afferent or returning limb of the loop is situated anterior to and eventually to the right of the efferent or outgoing limb Generally the terminal segment of the loop is written farther to the right than the initial deflection and it may even extend posteriorly Sometimes the QRS sÊ loop has a figure-of eight configuration in the horizontal projection in which case the first half of the loop is inscribed in a counterclockwise direction and the second *half in a clockwise direction*

b) Right sagittal QRS loop —The QRS sÊ loop in this projection is usually written inferiorly and anteriorly along roughly the +70 to +80 axis It may be written in a clockwise direction just as normally but more often it has a figure-of-eight contour the distal loop of which is inscribed in a counterclockwise direction

c) Frontal QRS loop —The frontal projection of the QRS sÊ loop lies for the most part to the left of the midline and is usually situated *within the* +30 *to* +90 segment of the frontal reference frame The loop is clockwise inscribed in most instances and exhibits a rightward and sometimes superiorly oriented terminal deflection

S–T Vector and T sÊ Loop

(See discussion in Chapter 8 of ventricular hypertrophy)

In the absence of digitalis effect an S–T vector is relatively uncommon in the vectorcardiographic RSR pattern of right ventricular hypertrophy although not infrequently an S–T vector is observed in the tall R pattern In the latter pattern the S–T vector can be ascribed in part at least to secondary repolarization changes consequent to the augmented depolarization forces produced by the right ventricle When present in right ventricular hypertrophy the S–T vector tends to be directed to the right slightly posteriorly and either superiorly or inferiorly

The most consistent feature of the T sÊ loop in either type of vectorcardiographic right ventricular hypertrophy pattern is that it is almost invariably discordant to the terminal mean instantaneous QRS vectors (Figs 103–107)

Combined Ventricular Hypertrophy, Ventricular Hypertrophy in Children

COMBINED VENTRICULAR HYPERTROPHY

Electrocardiographic Diagnosis

THE FREQUENCY with which significant hypertrophy of both ventricles is observed clinically and at post mortem has evoked numerous attempts in the past to define criteria for the electrocardiographic diagnosis of this condition That the electrocardiographic diag nosis of combined ventricular hypertrophy—if such a diagnosis can be made with any degree of certainty—is very tenuous can be inferred from the wide vari ety of criteria which have been proposed A resume of some of the findings thought by different authorities to be indicative of combined hypertrophy follows

CRITERIA OF COSBY AND ASSOCIATES—1 There is no significant reversal of the R/S ratio in V_1 but the onset of the intrinsicoid deflection in general occurs later than normally in this lead but not later than its onset in V_6

2 The R wave of V_1 and/or the R wave of V_6 exceeds the normal amplitude

LEPESCHKIN S CRITERIA—1 Right ventricular dila tation accompanying left ventricular hypertrophy may cause the precordial QRS transitional point to shift far to the left up to or beyond V_6 but the pattern of left ventricular strain* persists in the limb leads A second suggestive finding consists of the presence of an S wave in lead I with a left ventricular strain pat tern in the limb leads

2 Combined right and left ventricular hypertro phy may produce a tall R wave followed by an in verted T wave in right and left ventricular leads with a delayed intrinsicoid deflection in leads V_1 and V_6

3 If there is a perfect balance of potentials from the two hypertrophied ventricles the electrocardio gram may be normal

CRITERIA OF SODI PALLARES AND ASSOCIATES—1 A tall R wave may be present in lead V_1 with small or absent negative deflections The T waves in right precordial leads may be upright or inverted

2 Leads II III aVF V_5 and V_6 display tall peaked symmetrical T waves elevation of the S-T segments which are upwardly concave and tall R waves which in lead V_6 have delayed onset of the in trinsicoid deflection

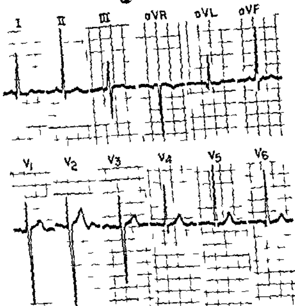
3 In combined ventricular hypertrophy due to ventricular septal defect Sodi Pallares and his asso ciates have observed large diphasic complexes in leads V V_3 and V_4 (Katz Wachtel sign) persistent S waves in V_5 and V_6 and a great shift to the right or upward of Â QRS

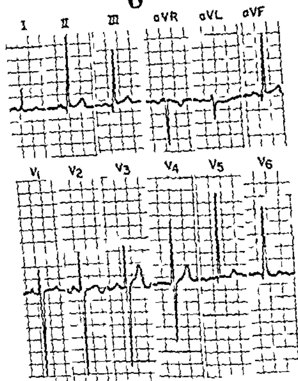
CRITERIA OF PAGNONI AND GOODWIN—1 A find ing which is considered suggestive of combined ven tricular hypertrophy is the association of electrocar diographic vertical heart position with signs of left ventricular hypertrophy such as delayed onset of the intrinsicoid deflection in lead V_5 (0 05 second or longer after onset of the QRS interval) and S-T seg ment depression and T wave inversion in the same lead

2 There are present an R wave greater than the Q wave in lead aVR an S wave larger than the R wave in V_5 and inversion of the T wave in V_1 to gether with signs of left ventricular hypertrophy

CRITERIA OF ROSENMAN AND KATZ—1 There is electrocardiographic evidence of left ventricular hy

*See the discussion of left and right ventricular strain on page 158





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pertrophy as defined in the criteria of Sokolow and Lyon

2 This is accompanied by indirect evidence of right ventricular hypertrophy such as clockwise rotation electrical heart positions other than horizontal or semihorizontal or the P mitrale pattern of atrial hypertrophy and/or atrial fibrillation

Vectorcardiographic Findings

Grishman and his associates have not reported observing any vectorcardiographic pattern pathognomonic of combined ventricular hypertrophy In certain forms of congenital heart disease which typically lead to biventricular hypertrophy (e g interventricular septal defects) these investigators have found vectorcardiographic manifestations of right ventricular hypertrophy in some cases and of left ventricular hypertrophy in others However in a considerable number of cases the vectorcardiogram ...

... ombined ventricular hypertrophy

THE VCG FINDINGS OF WHIPPLE COSIO AND LEVINE —In the horizontal projection of the vectorcardiogram recorded with the cube system of electrode placement the QRS loop is long and very narrow its length being at least 6 times its width The QRS loop is oriented to the left but not more than 20 posteriorly

THE VCG FINDINGS OF RICHMAN AND WOLFF —

1 The QRS sÊ loop lies inferiorly slightly anteriorly and to the left

2 The initial QRS vectors are directed anteriorly to the right and slightly superiorly or inferiorly

3 The subsequent QRS vectors of the centrifugal or efferent limb are directed anteriorly inferiorly and to the left

4 The early QRS vectors of the centripetal or afferent limb point more to the left and less anteriorly and inferiorly than the preceding vectors

5 The late QRS vectors are directed anteriorly or posteriorly and superiorly and to the right

6 The horizontal and frontal projections of the QRS sÊ loop have figure of eight configurations with the proximal loop counterclockwise inscribed and the distal loop clockwise inscribed

7 The sagittal projection of the QRS sÊ loop has a figure of eight configuration with the proximal loop clockwise inscribed and the distal loop counterclockwise inscribed

8 The T vectors are directed inferiorly posteriorly and to the left or away from the terminal QRS vectors

9 The T loops are inscribed clockwise in the horizontal and frontal projections and counterclockwise in the sagittal projection

In recording their vectorcardiograms Richman and Wolff utilize the Duchosal Sulzer double-cube system of electrode placement which tends to magnify the vertical component of the cardiac vector Otherwise their results can be applied to vectorcardiograms recorded with Grishman s cube modification of Duchosal Sulzer s lead system

Although we have observed vectorcardiographic patterns similar to those described by Whipple Cosio and Levine and by Richman and Wolff we have not been able to correlate these patterns specifically with combined ventricular hypertrophy In fact the QRS sÊ loop pattern described by Richman and Wolff has been recorded by us more frequently in cases of interatrial septal defect (a lesion not associated with biventricular hypertrophy) than in cases of patent ductus arteriosus or ventricular septal defect which frequently produce anatomic hypertrophy of both ventricles

At present it would seem that both the electrocardiographic and the vectorcardiographic manifestations of combined ventricular hypertrophy require further delineation (Fig 108)

VENTRICULAR STRAIN PATTERNS

In the original descriptions of ventricular strain patterns the term *left ventricular strain* was applied to the pattern of left axis deviation in the bipolar limb leads and T wave inversion in leads I and/or II while right axis deviation with T wave inversion in leads II and III was called *right ventricular strain* To these patterns was later attached the inference that left ventricular strain was suggestive of left ventricular hypertrophy and right ventricular strain of right ventricular hypertrophy As the science of electrocardiography developed and the precordial leads were added the terminology of left and right ventricular strain was retained with various amendments and modifications At present left ventricular strain is applied by some to the findings of T wave inversion in leads I or II and in leads V_4 to V_6 with QRS complexes of normal size and configuration The right ventricular strain pattern has been defined by Goldberger as consisting of marked clockwise rotation of the heart so that leads V_1 through V_5 or V_6 show rS or RS deflections accompanied by T wave inversion Inverted T waves may also be present in leads aVL and aVF and in leads II and III

The present status of these two patterns can be summarized as follows

1 The early definitions of right and left ventricular strain patterns were based on the very limited information available some of which was subsequently shown to be erroneous

2 *At present there is no agreement as to* what constitutes these electrocardiographic patterns nor agreement as to their implications

3 There is agreement however that the term *strain* has unfortunate connotations which have no place in electrocardiography

4 The so-called "left ventricular strain pattern" can appear in such widely diversified conditions as for instance coronary artery disease myocarditis and digitalis effect Some argument can be raised as to whether the T wave inversion of the right ventricular strain pattern necessarily reflects changes in the right ventricle at all Thus posterior rotation of the QRS loop due to various causes with the T loop maintaining its normal position within 30 to 60 of the former can produce T wave inversion over much of the precordium

5 There seems to be a general trend toward abandoning entirely the terms right ventricular strain" and left ventricular strain

VENTRICULAR HYPERTROPHY IN INFANCY AND EARLY CHILDHOOD

ular hypertrophy the fact soon becomes apparent that these criteria pertain chiefly to adults and are quite unreliable in infants and young children The factors responsible for the failure of the diagnostic criteria of left and right ventricular hypertrophy in the younger age group are primarily two (1) The

electrocardiogram and the criteria for the diagnosis of ventricular hypertrophy in this age group will be described

The Normal Vectorcardiogram and Electrocardiogram

The concept of *physiologic right ventricular preponderance* during the neonatal period is well sup

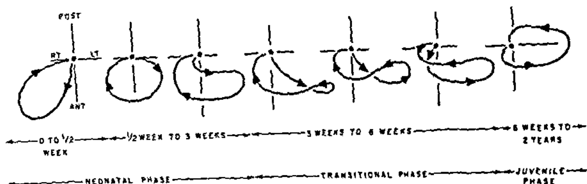


Fig 109 —A series of horizontal QRS loop …

electrocardiographic criteria for the diagnosis of right and left ventricular hypertrophy are based for the *most* part, on data derived from studies of adults (2) The normal electrocardiogram in inf…

… oltage of the QRS deflections in the left precordial leads or in all twelve leads of the routine electrocardiogram (*b*) relatively tall R waves in the right precordial leads (*c*) right axis deviation of the mean manifest electrical axis of QRS and (*d*) abnormally wide angular divergence of the mean QRS and T spatial vectors

Although vectorcardiographic studies of infants and young children are relatively limited in number to date there is reason to believe that the vectorcardiogram will prove a useful adjunct to the electrocardiogram in the diagnosis of ventricular hypertrophy in this age group

After the discussion of the vectorcardiographic findings in infants and young children the normal

ported by anatomic and electrocardiographic studies of the infant heart Moreover this concept is quite compatible with the fact that the fetal circulation burdens the right ventricle more than the left With the birth of the infant the left ventricle normally begins its lifelong performance of the greater proportion of the cardiac work at birth the weight of the right ventricle usually exceeds that of the left ventricle but after 2 weeks the left ventricle becomes slightly heavier than the right After 4–6 weeks the left ventricle begins to grow more rapidly than the right and within 7–12 months after birth the normal adult ratio of left ventricular weight to right ventricular weight is attained (approximately 2 1) There is preliminary evidence to indicate that the changing ratio of ventricular weights during the 12 month period following birth produces a characteristic sequence of vectorcardiographic changes This sequence will be described in three phases the neonatal transitional and juvenile phases (Fig 109)

For purposes of description the neonatal phase is

2 *The initial QRS vectors are directed anteriorly to the right and slightly superiorly or inferiorly*

3 The subsequent QRS vectors *of the centrifugal* or efferent limb are directed anteriorly *inferiorly, and* to the left

4 *The early QRS vectors of the centripetal or af*ferent limb point more to the left and less anteriorly *and inferiorly than the preceding vectors*

5 The late QRS vectors are directed anteriorly *or* posteriorly and superiorly and to the right

6 The horizontal and frontal projections of the QRS sE loop have figure of eight configurations with the proximal loop counterclockwise inscribed and the distal loop clockwise inscribed

7 The sagittal projection of the QRS sE loop has a figure of eight configuration with the proximal loop *clockwise inscribed and the distal loop counterclock*wise inscribed

8 The T vectors are directed *inferiorly posteri*orly and to the left or away *from the terminal QRS* vectors

9 The T loops are inscribed clockwise in the horizontal and frontal projections and counterclockwise in the sagittal projection

In recording their *vectorcardiograms Richman* and Wolff utilize the Duchosal Sulzer double cube *system of electrode placement which tends to mag*nify the vertical component of the cardiac vector *Otherwise their results can be applied to vectorcardio*grams recorded with Grishman s cube modification of Duchosal Sulzer s lead system

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5 There seems to be a general trend toward abandoning entirely *the terms right ventricular strain"* and "left ventricular strain

loops are typified by a figure-of-eight configuration and an anterior orientation At first the distal loop of the eight" is small because only a very small initial segment of the afferent limb lies relatively posterior to the efferent limb Later more and more of the afferent limb of the QRS loop comes to lie behind the efferent limb until late in the transitional period the loop is written almost entirely in a counterclockwise direction to the right and anteriorly or slightly posteriorly

RIGHT SAGITTAL QRS LOOP —The loop is usually situated anteriorly and inferiorly and has a clockwise direction of inscription

FRONTAL QRS LOOP —Generally the frontal loops are oriented mainly inferiorly and to the left and are written in a clockwise direction They may display a terminal deflection to the right and superiorly

The horizontal QRS loops described above may project R waves with slurred or notched upstrokes or rSR complexes on lead V_1

JUVENILE PHASE

HORIZONTAL QRS LOOP —Occasionally the QRS loop has a figure-of-eight configuration but more often it is written entirely in a counterclockwise direction In general the loop tends to resemble the normal adult horizontal loop except for the following features (*a*) the loop lies either entirely *anteriorly* or half anteriorly and half posteriorly and (*b*) not infrequently the loop exhibits a terminal appendage (without conduction delay) directed to the right and posteriorly

RIGHT SAGITTAL QRS LOOP —The major portion of the loop usually lies inferiorly and either entirely anteriorly or half anteriorly and half posteriorly It is written in a clockwise direction

FRONTAL QRS LOOP —Usually the QRS loop is oriented to the left and inferiorly and is inscribed in a clockwise direction

If the horizontal QRS loop lies entirely anteriorly lead V_1 tends to register an R wave of greater amplitude than the following S wave If the long axis of the loop parallels the positive axis of lead V_6 there may appear in the precordial leads the electrocardiographic pattern of counterclockwise rotation " an RS deflection being written in V_2 and resultantly nor[illegible]

complex [illegible] may record an RSr′

The upper age limit for the juvenile type of vectorcardiographic pattern is not well defined Apparently horizontal QRS loops tending to display a more anterior orientation than the loops of normal adults have been recorded in children 5 years of age Lamb and Dimond who utilize their own system of electrode placement rather than the cube system have observed horizontal QRS loops having a figure-of-

TABLE 9 —NORMAL STANDARDS FOR THE R S * AND T† WAVES IN CHILDREN ACCORDING TO AGE GROUP‡

	NEWBORN TO 1 YEAR	1–10 YEARS	10–20 YEARS	20 YEARS AND OVER
Lead aVR				
R—Maximum	9 0	6.5	8 0	4 1
Mean	2.32	1 6	1.39	0 91
Lead aVL				
R—Maximum	10 0	11 8	10 1	10 1
Mean	3 33	3 15	2 21	2 61
Lead V_4				
R—Maximum		9 9	8 0	
Mean		4 18	3 8	
Lead V_1				
R—Minimum	3 0	0 4	0 4	0
Maximum	29	20 0	16 7	15 5
Mean	13 61	7 15	5.29	3 09
S—Minimum	0	0	0	0 8
Maximum	28 0	38.5	26 6	26.2
Mean	8.57	11 02	11 09	9 41
Lead V				
R—Minimum	0	5 0	3.5	2 0
Maximum	24 0	23 0	25 0	22 6
Mean	8 0	11 91	11 11	9 68

T WAVES	V	V_4	V_3	V	V_6	V
Inverted	16 yr	12 yr	10 yr	5 yr	15 hr	8 hr
Diphasi	16 yr	18 yr	15 yr	11 yr	14 hr	1 day

* The R and S waves are considered in terms of their minimum maximum, and mean amplitude values (expressed in millimeters) for each age group in the scalar leads used to diagnose left and right ventricular hypertrophy

†The T waves are described in terms of the average upper age limit at which they may be found to be inverted

‡ [illegible] and Criteria for Diagnosis of Diseases of the Heart and Blood Vessels ([illegible] ed. New York Heart Association 19 3) the data were compiled from authoritative sources

eight configuration commonly in children up to 6 years of age but only rarely in normal adults over 20 years It is evident that studies of larger series of infants and children are a necessity However several tentative conclusions seem warranted

1 In the diagnosis of right ventricular hypertro-

here defined as the sequence of changes occurring during the first 3 weeks after birth, the *transitional phase* during the second 3 weeks (fourth through sixth postnatal week), and the *juvenile phase* from the age of 6 weeks to 2 years or older. Obviously, the age groups included in these phases are subject to future change as more information about the normal vectorcardiogram in the young is accumulated.

Neonatal Phase

Horizontal QRS loop.—Early in this period the QRS sÊ loop lies almost entirely anteriorly and to the right and is inscribed in a clockwise direction (Fig 109). Later, the initial deflection and early portion of the loop are written to the left, and then to the left and anteriorly; the afferent limb passes anteriorly and to the right before returning to the point of origin. Late in the neonatal period the QRS loop tends to lie more to the left than to the right. It displays an initial deflection to the right and anteriorly, and then the efferent limb of the loop is written somewhat anteriorly but more to the left than previously. Finally, the *afferent limb is inscribed in a clockwise direction to the right and anteriorly*, as illustrated in Figure 110.

Right sagittal QRS loop.—The neonatal phase is characterized by an anteriorly oriented sagittal loop which usually, although not invariably, is written in a clockwise direction.

Frontal QRS loop.—Early in this phase the loop lies essentially to the right and inferiorly and is written in a counterclockwise direction. Subsequently the frontal QRS loop tends to be located less to the right and more to the left and is written in a clockwise direction.

The vectorcardiographic patterns described above, if recorded from adults, would be considered typical of right ventricular hypertrophy, as would the electrocardiographic findings. Thus, the first horizontal loop pattern described above projects a tall R wave on V_1, the second a qR complex, and the third loop configuration an rR or rSR complex.

Transitional Phase

Horizontal QRS loop.—This projection best illustrates the transition from the neonatal loop configuration of physiologic right ventricular preponderance to the loop configuration observed in children 11 months of age and older. The transitional QRS



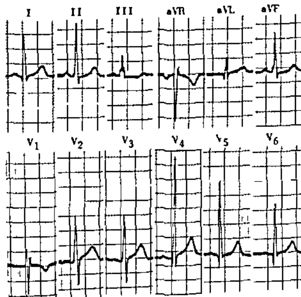


Fig 110.—Normal electrocardio- [illegible] limb and precordial leads and the RSR deflection in lead V. In the vectorcardiogram, the directions of inscription of the horizontal and sagittal QRS loops are normal except for the terminal portion of each loop, which is clockwise inscribed in the horizontal projection and counterclockwise inscribed in the sagittal projection. Both efferent and afferent limbs of the QRS sÊ loop are displaced anteriorly, the former more than the latter, in contrast to the adult pattern of right ventricular hypertrophy. Note also the prominent rightward, anterior and inferior deflection of the early part of the QRS sÊ loop.

phy in infants and young children the electrocardiograph has obvious limitations

2 It may be impossible to distinguish *pathologic* from *physiologic* right ventricular preponderance in the vectorcardiograms of infants less than 31 days old

3 However the diagnosis of pathologic right ventricular preponderance can probably be made in such infants by recording serial vectorcardiograms According to Elek and his associates the transitional vectorcardiographic pattern usually appears by the time an infant is 31 days old or shortly thereafter If serial vectorcardiograms fail to demonstrate the normal sequence of changes in the vectorcardiogram culminating in the appearance of the juvenile pattern then pathologic right ventricular hypertrophy is probably present

The sequence of vectorcardiographic patterns described above is compatible with the electrocardiographic studies of Ziegler in infants and young children of corresponding age groups Ziegler found that the average amplitude of the R wave in lead V_1 exceeds that in V_6 from birth to approximately 6 months of age that the two amplitudes are nearly equal in

...

gressively throughout infancy and childhood, although even at age 16 the amplitude of the R wave is slightly greater than that in lead V_1 of the average normal adult Within the period of 6 months to 3 *years the R S ratio in* V_1 becomes on the average less than 1 Table 9 shows according to age group the normal maximum minimum and mean values for the R and or S waves in the electrocardiographic leads of chief diagnostic importance in right and left ventricular hypertrophy See also Figures 111 and 112 for normal electrocardiographic and vectorcardiographic findings

The ECG Diagnosis of Right Ventricular Hypertrophy

It is not possible to list *all* of the many electrocardiographic criteria which have been p...

... of Goodwin are widely used and are summarized below

Criteria of Sokolow and Lyon —1 In lead aVR the R wave has an amplitude of 5 *mm* or more

2 Lead V_1 is characterized by an R wave amplitude of 7 mm or more an S wave depth of 2 mm or less a T wave inverted and preceded by an R wave 5 mm or more in amplitude an R/S ratio greater than 4 in children under 5 years and greater than 1 in children over 5 years and ventricular activation time or pre intrinsicoid deflection time of 0 04 second or longer

3 Lead V_5 shows an R wave amplitude of 4 mm or less and an R/S ratio of 1 or less

4 In leads V_1 and V_5 $R_{V_1} + S_{V_5} = 10.5$ *mm* or more in children over the age of 5 years and

$$\frac{\text{R/S ratio in } V_5}{\text{R/S ratio in } V_1} = 0.04 \text{ or less}$$

5 In lead V_6 the S wave has a depth of 7 mm or more

Goodwin's criteria —1 In leads V_{3R} and V_1 the qR complex has a Q/R ratio less than 1 or the Rs complex has an R/S ratio greater than 1 the ventricular activation times in both types of QRS configuration exceeding 0 03 second

2 In lead aVR the Q/R ratio is less *than 1 or the* ventricular activation time is 0 06 second *or more*

...

...ocardiographic criteria for right ventricular hypertrophy proposed by Goodwin and by Sokolow and Lyon were evaluated It was found that Goodwin's criteria failed diagnostically *in a significant percent*age of the patients with right ventricular hypertrophy The criteria of Sokolow and Lyon were fulfilled in virtually all the electrocardiograms of patients with right ventricular hypertrophy However Braunwald and his associates pointed out that application of these criteria to the large series of normal infants and children studied by Ziegler would result *in many* of these normals *meeting the diagnostic* requirements for right ventricular hypertrophy *It* was the opinion of these investigators that the following approach to the electrocardiographic diagnosis of right ventricular hypertrophy may prove *the most satisf*...

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in V_6 exceed ... the normal maximum values in Table 9 (*b*) the S wave in V_1 and the R in V_6 display ...

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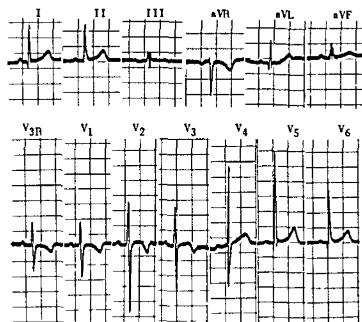
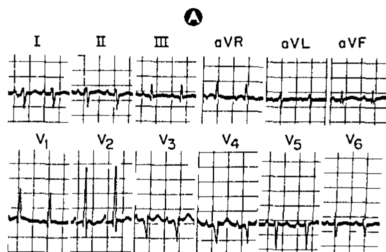
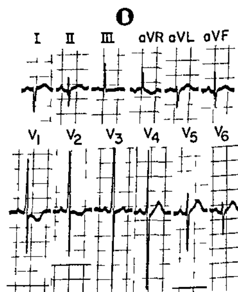


Fig 111 —Normal electrocardiogram and vectorcardiogram in a boy 4 without heart disease Note in the electrocardiogram the almost equiphasic RS deflection in lead V_1 and the inverted T waves extending through lead V_3 These findings are normal for this age group In the vectorcardiogram the QRS sÊ loop displays a *prominent anterior and rightward early deflection* which corresponds to the relatively tall R wave in lead V_1 of the electrocardiogram





[illegible] old premature boy There are marked right [illegible] al QRS transition This electrocardiogram in [illegible] s normal in a neonatal infant The findings probably reflect persistence of the *physiologic right ventricular preponderance* normally present in the fetus B normal electrocardiogram in girl 1 month also displaying the findings of physiologic right ventricular preponderance

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[illegible] ratio in V_1 becomes on the average less than 1 Table 9 shows according to age groups the norm [illegible]
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2 In lead aVR the Q/R ratio is less than 1 or the ventricular activation time is 0 06 *second or more*

CRITERIA OF BRAUNWALD AND ASSOCIATES —Braunwald and his co-workers have studied very thoroughly a large series of patients with congenital heart disease the patients ranging in age from under 3 years to over 30 years (only two were less than 3 *years old*) The electrocardiographic criteria for right ventricular hypertrophy proposed by Goodwin and by Sokolow and Lyon were evaluated It was found that Goodwin's criteria failed diagnostically in a significant percentage of the patients with right ventricular hypertrophy The criteria of Sokolow and Lyon were fulfilled in virtually all the electrocardiograms of patients with right ventricular hypertrophy However Braunwald and his associates pointed out that application of these criteria to the large series of normal infants and children studied by Ziegler would result in many of these normals meeting the diagnostic requirements for right ventricular hypertrophy It was the opinion of these investigators that the following approach to the electrocardiographic diagnosis of right ventricular hypertrophy may prove the most satisfactory

Electrocardiographic findings diagnostic of right ventricular hypertrophy—Right ventricular hypertrophy is almost certainly present if the electrocardiogram (1) satisfies the criteria of Sokolow and Lyon and (2) meets the following requirements (*a*) the R waves in leads aVR V_{3R} and V_1 and the S waves in V_6 exceed in amplitude the normal maximum values in Table 9 (*b*) the S wave in V_1 and the R wave in V_6 display voltages less than the [illegible]

ular activation time in V_1 meets the requirements of 0 04 second or more

Electrocardiographic findings suggestive of right ventricular hypertrophy —If the electrocardiogram meets only the requirements of Sokolow and Lyon it can be considered merely suggestive of right ventricular hypertrophy and vectorcardiographic studies should be performed

Electrocardiographic findings rendering the diagnosis of right ventricular hypertrophy unlikely —If the electrocardiogram does not fulfill any of the above requirements predominant right ventricular hypertrophy secondary to congenital heart disease is almost certainly ruled out

RSR complex in V_1 —The preceding statements cannot be applied to electrocardiograms showing an RSR complex in lead V_1 with or without prolonged intraventricular conduction It is in the clarification of electrocardiographic patterns such as this that the vectorcardiograph proves its diagnostic value For example right ventricular hypertrophy incomplete right bundle branch block and late depolarization of the pulmonary conus region in a normal heart may all produce RSR complexes in lead V_1 which may be quite difficult if not impossible to distinguish electrocardiographically one from another However the vectorcardiographic patterns of right ventricular hypertrophy and of incomplete right bundle branch block can be readily differentiated Moreover it is possible to recognize as a normal variant the QRS loop which has a terminal deflection to the right and posteriorly and which produces a small terminal R wave in V_1 in the absence of right ventricular hypertrophy and right bundle branch block presumably as the result of late activation of the pulmonary conus region

The ECG Diagnosis of Left Ventricular Hypertrophy

Braunwald and his associates in their series of children and young adults with congenital heart disease who ranged in age from 3 to over 30 years evaluated the criteria of Sokolow and Lyon for the electrocardiographic diagnosis of left ventricular hypertrophy (see Chapter 9) They also utilized the amplitude standards which appear in Table 9 These investigators found that the requirements proposed by Sokolow and Lyon were met by about 54% of the patients with left ventricle hypertrophy while only 32% of the patients presented electrocardiograms outside the limits of normal defined in Table 9 Thus in children as well as in adults the electrocardiographic diagnosis of left ventricle hypertrophy is undoubtedly marred by a significant percentage of false negative and false positive interpretations

Congenital Heart Disease, Mitral Stenosis, and Cor Pulmonale General Considerations

CLINICALLY right ventricular hypertrophy occurs primarily in three types of heart disease congenital heart disease mitral stenosis and chronic cor pulmonale Tl e f ... d -

... of heart disease but only recently has attention been called to the differences existing in the prominence of the electrocardiographic and vectorcardiographic manifestations of right ventricular hypertrophy in these conditions

To illustrate the trend in the electrocardiographic findings of right ventricular hypertrophy in mitral stenosis in chronic cor pulmonale and in congenital heart disease the electrocardiograms of a number of patients with vectorcardiographically diagnosed right ventricular hypertrophy (with postmortem or surgical confirmation in most of the mitral stenosis and congenital heart disease cases) were reviewed by us (see Table 10) to determine the following points

1 The number of electrocardiograms in each of the above three groups in which diagnosis of right ventricular hypertrophy could be made using—
   a) The criteria of Barker and Valencia for the diagnosis of right ventricular hypertrophy with complete and incomplete right bundle branch block—an R deflection in V exceeding 15 mm in complete right bundle branch block and an R deflection exceeding 10 mm in incomplete right bundle branch block
   b) The criteria of an R/S amplitude ratio in V_1 greater than 1 and an R wave in V of 7 mm or more amplitude in the absence of an RSR deflection in V_1
2 The average amplitude of the R wave or R wave of an RSR deflection in lead V in the electrocardiograms
3 The number of cases in the three groups in which the vectorcardiograms were diagnostic of right ventricular hypertrophy and the frequency of each of the two vectorcardiographic patterns of right ventricular hypertrophy in these cases

Attention must be called to the fact that the data presented below have no precise statistical significance because of the manner in which the patients were selected for study

Table 10 does not present any statistically valid data as to the relative frequency of the electrocardiographic and vectorcardiographic findings of right ventricular hypertrophy in congenital heart disease mitral stenosis and chronic cor pulmonale However as the table indicates there is ample evidence attesting to the lower incidence of these findings in mitral stenosis and their relative rarity in chronic cor pulmonale Both the frequency of occurrence and the prominence of the electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy depend in all probability on such factors as the following

1 Factors affecting the manner in which the QRS forces are transmitted to and recorded by the lead electrodes
   a) The conductivity of the intrathoracic structures
   b) Cardiac position and rotation and the location of the electrical center of the heart or electrical null point
2 The duration degree and type of right ventricular overloading (whether systolic diastolic or both—i e composite overloading)
3 The presence or absence of coexisting left ventricular overloading

Each of the above factors will be considered in some detail in the following paragraphs

TABLE 10.—COMPARISON OF ECG AND VCG FINDINGS OF RIGHT VENTRICULAR HYPERTROPHY IN CONGENITAL HEART DISEASE, MITRAL STENOSIS, AND CHRONIC COR PULMONALE

	CONGENITAL HEART DISEASE*	MITRAL STENOSIS†	CHRONIC COR PULMONALE‡
Total number of cases	50	53	19
Cases with ECG right bundle branch block	2	0	0
R′ > 15 mm in V_1	0	0	0
Av. R′ amplitude in V_1	13 mm		
VCG right ventricular hypertrophy (+ or − right bundle branch block)	2	0	0
Cases with RSR′ in V_1 less than 0.11 sec. duration	14	28	5
R′ > 10 mm in V_1	7	0	0
Av. R′ amplitude in V_1	13 mm	5 mm	3 mm
VCG right ventricular hypertrophy	13	15	3
Cases with *R/S amplitude ratio in* V_1 > 1	26	18	0
R in $V_1 \geqq 7$ mm	25	9	0
R in $V_1 < 7$ mm	1	9	0
Av. R amplitude in V_1	19 mm	8 mm	
VCG right ventricular hypertrophy	26	15	0
Cases with R/S amplitude ratio in $V_1 < 1$	8	7	14
VCG right ventricular hypertrophy	0	2	1
Total number of cases with VCG right ventricular hypertrophy	41	32	4
VCG tall R QRS sÊ loop pattern of right ventricular *hypertrophy*	21	7	3
VCG RSR′ QRS sÊ loop pattern of right ventricular hypertrophy	18	25	1
VCG right ventricular hypertrophy and right bundle branch block	2	0	0

*Includes only those congenital cardiac anomalies which with varying frequency produce right ventricular hypertrophy.

†Includes cases of pure mitral stenosis and cases of hemodynamically predominant mitral stenosis with mitral insufficiency and/or aortic valve disease.

‡Diagnosis was made on the basis of either postmortem proof of right ventricular hypertrophy or unequivocal radiologic evidence of chronic cor pulmonale.

FACTORS AFFECTING THE FREQUENCY AND PROMINENCE OF THE PATTERNS OF RIGHT VENTRICULAR HYPERTROPHY

Manner of Transmission of QRS Forces to the Lead Electrode

The transmission of QRS forces to the body surface electrodes is influenced by factors such as those to be described in the following paragraphs.

Conductivity of intrathoracic structures.—The influence of this factor can be illustrated by the following examples.

1. *Chronic pulmonary emphysema* is responsible for virtually all cases of chronic pulmonary heart disease with right ventricular hypertrophy (chronic cor pulmonale). In pulmonary emphysema, the walls of

many of the alveoli in the lungs are ruptured and the affected alveoli coalesce to form abnormally large air sacs The latter remain overdistended with air be cause of the poor respiratory exchange Thus the lungs contain more air than normally and since air is a poor conductor the electrical forces produced by the heart are not transmitted to the chest electrode as well as normally As a result, there may be low voltage of the electrocardiographic deflections in all leads despite the presence of right ventricular hyper trophy although this feature may be more marked in some leads than in others because of variations in the amount of air filled lung between heart and electrode

2 Rather marked degrees of right atrial dilatation are noted more commonly in congenital heart diseases than in mitral stenosis or pulmonary heart disease If it is true as some authorities believe that right atrial dilatation facilitates transmission of the forces produced in the hypertrophied basal right ventricular wall and septum then this factor may well play some part in determining the higher incidence of prominent right ventricular hypertrophy patterns in electrocardiograms of patients with congenital heart disease than in those of patients with acquired heart disease

Cardiac position rotation and the location of electrical center of the heart—The following examples illustrate the influence of this factor

1 In congenital heart disease with pulmonic stenosis the long axis of the heart is believed by some investigators to be more horizontal than in any other type of right ventricular hypertrophy As a result the basal wall and outflow tract of the right ventricle (crista supraventricularis) and basal septum (the regions which undergo maximum hypertrophy in right ventricular hypertrophy) are oriented toward the positive half of the axis of derivation of lead V_1 Hence right precordial leads in this instance optimally record the augmented forces arising in the hypertrophied right ventricle

2 In chronic cor pulmonale according to Sodi Pallares the heart is located much lower in the chest than in either normal subjects or those with congenital or mitral stenotic heart disease In addition the heart is rotated clockwise on its longitudinal axis and its apex is displaced posteriorly Sodi Pallares believes that, because of the lower position of the heart in the chest, the precordial electrode explores the atria rather than the hypertrophied right ventricle He also postulates that the clockwise rotation of the heart displaces the left ventricle posteriorly thereby causing all six precordial leads of the electrocardiogram to display low R waves with relatively deep S waves even though there may be anatomic right ventricular hypertrophy As one might infer from the preceding explanation Sodi Pallares considers the electrocardiographic precordial leads to be semidirect leads which respond to proximity potentials arising in myocardium closest to the exploring electrode

The frequent failure of the electrocardiogram and vectorcardiogram to present diagnostic evidence of right ventricular hypertrophy in chronic cor pulmonale can perhaps be explained somewhat differently in terms of the vector or equivalent dipole concept It is probable that the marked changes in the anatomic position and rotation of the heart in chronic pulmonary emphysema are accompanied by a shift in the location of the electrical center of the heart Since the effective axis of a given lead or the direction of the lead vector is a function of the location of the dipole center its eccentricity etc a change in the site of the dipole center might be expected to alter the manner in which the lead in question responds to the transverse sagittal or vertical components of the cardiac vector Thus it is conceivable that a lead with an anteroposterior anatomic axis might have an effective axis which is so tilted that instantaneous vectors situated anteriorly are recorded by the lead as if they were located posteriorly At present such an explanation is purely conjectural although certain observations which we have made (described later) provide some evidence to support this hypothesis

Duration Degree and Type of Right Ventricular Overloading

In congenital heart disease as a general rule right ventric[illegible]

[illegible] however on the average the hemodynamic burden imposed on the right ventricle is likely to be greater in congenital than in acquired cardiac lesions

In order to interrelate better the anatomic, hemodynamic and electrocardiographic changes accompanying the various congenital and acquired cardiac lesions Cabrera and Monroy have categorized the latter according to whether a given cardiac lesion produces systolic diastolic or composite o[illegible] of the left [illegible] ing of affecte[illegible] [illegible] as the result of an increased resistance to the ejection of blood during systole Systolic overloading typically leads to concentric hypertrophy (i e muscular hypertrophy un

TABLE 10 – COMPARISON OF ECG AND VCG FINDINGS OF RIGHT VENTRICULAR HYPERTROPHY IN CONGENITAL HEART DISEASE, MITRAL STENOSIS AND CHRONIC COR PULMONALE

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R in V_1 ≥ 7 mm	25	9	0
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*Includes only those congenital cardiac anomalies which with varying frequency produce right ventricular hypertrophy.
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FACTORS AFFECTING THE FREQUENCY AND PROMINENCE OF THE PATTERNS OF RIGHT VENTRICULAR HYPERTROPHY

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Congenital Heart Disease

Although the clinician is quite cognizant of the fact that electrocardiography is not a substitute for angiocardiography and cardiac catheterization in the diagnostic study of congenital heart disease he probably is less aware of or tends to underestimate the diagnostic value of the electrocardiogram and vectorcardiogram in this type of cardiac disease Admittedly the information provided by the electrocardiogram and vectorcardiogram is relatively nonspecific in most instances Nevertheless if used properly it often has significant diagnostic and prognostic value Moreover complex diagnostic procedures like angiocardiography and cardiac catheterization ordinarily are not available to the physician who first examines a patient suspected of having congenital heart disease and yet the physician must make the decision as to whether or not this diagnosis is a likely enough possibility to merit further study Electrocardiography on the other hand is a diagnostic procedure which is readily available to most physicians and one that is easily performed

The purpose of this chapter is to review briefly the anatomic and hemodynamic characteristics of certain congenital heart diseases and then to relate them to the electrocardiogram and vectorcardiogram When possible the clinical implications of the electrocardiographic and vectorcardiographic findings will be cited Since the number of cases of congenital cardiac anomalies which we studied was small (see Table 11) the findings will be supplemented with addi

TABLE 11 —Cases of Congenital Heart Disease Studied by the Authors

Type of Congenital Heart Disease	No of Cases ECG	VCG
Isolated pulmonic stenosis with normal aortic root	8	5
Interatrial septal defect	15	14
Interventricular septal defect	9	4
Patent ductus arteriosus	11	5
Tetralogy of Fallot (or pentalogy)	9	4

Postmortem or surgical confirmation of the diagnosis was available in almost all cases.

[illegible]

cause of their relatively greater frequency of occurrence and or amenability to surgical correction

TYPES OF CONGENITAL HEART DISEASE

The following classification emphasizes the hemodynamic effects of the cardiac abnormality

I Stenotic lesions causing systolic overloading of the right or left ventricle
  a) Overloading of the left ventricle
    1 Coarctation of the aorta (adult type with closed ductus arteriosus)
    [illegible]
  b) [illegible]

II [illegible]

  2 Patent ductus arteriosus with left to right shunt
  b) Diastolic overloading of the left ventricle and systolic overloading of the right ventricle
    1 Interventricular septal defect with right to-left

accompanied by chamber dilatation) When there is *diastolic overloading* of one or the other ventricle the volume of blood in the affected ventricle during diastole greatly exceeds that present normally This causes eccentric hypertrophy of the affected ventricle which is characterized by a lesser degree of muscular hypertrophy than in *systolic overloading* but the hypertrophy is accompanied by dilatation of the affected chamber *Composite overloading* is the term used when both systolic and diastolic overloading of one or the other ventricle are present Cabrera and Monroy s concept of ventricular overloading resembles the classification of *right ventricular strain* proposed by Donzelot and his associates Thus systolic overloading *corresponds to the term of Donzelot and his* co workers *hypertrophy by barrier* diastolic overloading to their *hypertrophy by overload* (or shunt) and composite overloading is in essence what they designate as *hypertrophy by adaptation*

Although the concept of ventricular overloading and the related concept of hypertrophy by barrier overload and adaptation have a certain degree of validity, nevertheless Braunwald and his associates in Grishman s laboratory the authors of this text and other investigators have not noted as consistent a correlation of electrocardiographic findings with the type of congenital or acquired cardiac disease present as have been reported by Cabrera and Monroy and by Donzelot and his associates An additional fact worthy of mention is that in all probability a high percentage of the incomplete right bundle branch block patterns observed by Cabrera and Monroy in cases with diastolic overloading of the right ventricle were actually right ventricular hypertrophy patterns of the RSR′ type This much is true however congenital or acquired *cardiac lesions which cause systolic overloading* of the left or right ventricle tend to be accompanied by concentric hypertrophy with marked thickening of the muscular wall of the affected ventricle On the other hand, lesions which lead to diastolic overloading of the left or right ventricle are associated primarily with ventricular dilatation and relatively minimal hypertrophy of the ventricular wall (*eccentric hypertrophy*) *In such instances* there may be selective hypertrophy of the crista supraventricularis and trabeculae of the right ventricle the electrical effects of which are much less prominent than the electrical effects of concentric hypertrophy

Presence or Absence of Coexisting Left Ventricular Overloading

In the acquired mitral and pulmonary types of heart disease, prior to onset of right ventricular overloading the left ventricle is almost invariably electrically predominant with reference to the right ventricle Consequently the electrical effects of anatomic right ventricular hypertrophy are, in varying degree offset by the initial preponderance of left ventricular forces If the anatomic right ventricular hypertrophy is *relatively minimal* its electrical effects may not be sufficient to overcome the normal predominance of the left

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incidence of electrocardiographic right ventricular hypertrophy in mitral stenosis than in congenital heart disease since many of the cyanotic congenital *anomalies such as tetralogy of Fallot* are associated with hypoplasia of the left ventricle In addition to the fact that mitral insufficiency which causes left ventricular overloading often coexists *with mitral* stenosis it must be remembered that in rheumatic heart disease there frequently is multivalvular and myocardial involvement For example mitral stenosis may be accompanied by aortic stenosis and/or insufficiency both of which produce left ventricular hypertrophy or diffuse myocardial scarring consequent to rheumatic carditis can lead to dilatation and *hypertrophy of the left ventricle In the presence* of left ventricular hypertrophy the degree of anatomic ... and therefore the de-

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manifestations of right ventricular hypertrophy than would otherwise be the case and—more often than not—the left ventricular hypertrophy prevents the appearance or obscures the electrocardiographic manifestations of right ventricular hypertrophy

The three types of heart disease—congenital heart disease mitral stenosis and cor pulmonale—will be discussed fully and separately in the following three chapters

of blood to satisfy the demands of the fetal circula

type of coarctation

arteriosus is unable to main

velops in utero in contrast with the situation in the infantile type

Typically in coarctation of the aorta there is hypertension in the upper extremities and weak or absent arterial pulsations in the lower extremities The hypertension is usually systolic although a varying

but two mechanisms have been entertained (a) some authorities attribute the hypertension to mechanical obstruction of blood flow by the coarctation itself while (b) others implicate renal ischemia secondary to the diminished blood flow to the kidneys There is evidence for and against both mechanisms In any event, depending on the degree of coarctation and the extent of the collateral circulation there is systolic overloading of the left ventricle whether this is attributable to the mechanical effects of the coarctation to hypertension of renal origin or to both factors In addition the hemodynamic burden imposed on the left ventricle is sometimes increased even further by associated cardiac malformations the most common of which is a bicuspid aortic valve In the presence of the latter anomaly aortic insufficiency can result from incompetence of the aortic cusps due to hypertension and to dilatation of the aorta proximal to the coarctation

In brief if there is marked stenosis of the aorta or if the collateral circulation is poorly developed coarctation of the aorta may produce severe systolic overloading of the left ventricle which in turn may cause concentric hypertrophy of the left ventricle On the other hand if the coarctation is of a relatively mild degree or the collateral circulation well developed there may be minimal left ventricular hypertrophy Obviously all degrees of anatomic left ventricular hypertrophy between the preceding two extremes may be encountered.

The ECG and VCG Findings

To provide a more representative sampling of the reported electrocardiographic findings in the adult type of coarctation of the aorta data regarding various series of coarctation cases reported by a number of investigators were pooled and the incidence of each of the principal electrocardiographic findings in coarctation was computed for the entire group collectively The series from which the data were obtained includes those reported by the following Braunwald Hjellberg Metianu and Oglesby and their collaborators and Landtman Ziegler and Sokolow and Edgar Their findings follow

1 The electrocardiogram was normal in approximately one third of the cases Braunwald and his associates report that normal vectorcardiograms were recorded in 4 of 11 cases of coarctation of the aorta

2 Left ventricular hypertrophy was observed in slightly over half of the cases By and large in most cases the electrocardiographic features of left ventricular hypertrophy were not particularly prominent It has been stated that the presence of marked evidence of left ventricular hypertrophy may be indicative of an associated aortic valvular lesion Left ventricular hypertrophy was diagnosed vectorcardiographically in 7 of the 11 cases referred to above Although Braunwald and his coworkers noted nothing unique about the vectorcardiographic pattern of left ventricular hypertrophy occurring in coarctation the authors of this text were impressed in the few cases they studied by the fact that the T sE loop was concordant rather than discordant to the QRS sE loop typical of the vectorcardiogram in left ventricular hypertrophy due to other cardiac lesions

3 The mean manifest electrical axis of QRS (mean frontal plane QRS vector) was normally situated in about 60% of the cases while about 30% of the cases showed left axis deviation and about 10% right axis deviation Although the incidence of left axis deviation is lower perhaps in coarctation of the aort

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several types of cardiac disease which can give rise to concentric hypertrophy of the left ventricle In all probability the reason that acquired valvu

shunt due to pulmonic stenosis or pulmonary hypertension (including Eisenmenger's complex)
2. Patent ductus arteriosus with right to left shunt due to pulmonary hypertension

c) Diastolic overloading of the right ventricle
1. Interatrial septal defect with left to right shunt (including Lutembacher's syndrome consisting of congenital mitral stenosis and an interatrial septal defect)

d) Diastolic and systolic (composite) overloading of the right ventricle
1. Interatrial septal defect with right-to-left shunt due either to pulmonic stenosis (trilogy of Fallot) or to pulmonary hypertension

III. Multiple cardiac anomalies associated with other developmental defects
a) Dextrocardia with and without situs inversus
b) Tetralogy of Fallot

According to Cabrera and Monroy, the electrocardiographic manifestations of systolic and diastolic overloading of the right and left ventricles are as follows:

I. Right ventricle*
a) Diastolic overloading
1. Complete or incomplete right bundle branch block
2. Right axis deviation

b) Systolic overloading
1. Increased voltage of the R wave in lead V_1 and sometimes initial slurring of the R wave in this lead
2. Inverted T wave in lead V_1 if systolic overloading of the right ventricle is marked and of long duration
3. The configuration of the QRS deflection in lead V_1 is usually monophasic or diphasic. However, if the systolic overloading is complicated by diastolic overloading, the QRS deflection may appear notched and polyphasic
4. Right axis deviation

II. Left ventricle*
a) Diastolic overloading
1. Tall R wave in V_5 and V_6 with delayed onset of the intrinsicoid deflection
2. Deep S wave in leads V_1 and V_2
3. Tall upright T wave in leads V_5 and V_6 without opposition between Â QRS and Â T

b) Systolic overloading
1. Inverted T waves and/or depressed S-T segments in left precordial leads
2. Opposition of Â QRS and Â T

STENOTIC LESIONS CAUSING SYSTOLIC LEFT VENTRICULAR OVERLOADING

Coarctation of the Aorta

Anatomically and hemodynamically, there are two types of coarctation of the aorta—the infantile type and the adult type.

In the *infantile type* there is a marked elongated narrowing of the aortic segment between the origin of the left subclavian artery and the opening of a patent ductus arteriosus. After birth, a fetal type of circulation (pulmonoaortic shunt) through the patent ductus persists, and the right ventricle has to meet the circulatory demands of both the pulmonary circulation and the systemic circulation distal to the coarctation. Thus, at birth the right ventricle is suddenly burdened with a highly pathologic circulation, and, with rare exceptions, severe heart failure and death ensue within several days. The exceptional patients apparently have some other cardiac malformation and intracardiac shunt in addition to the infantile coarctation, but even in these cases life seldom is prolonged beyond the juvenile period. In the few patients studied, the cardiac anomalies were found to be hemodynamically equivalent to a patent ductus arteriosus with a pulmonoaortic shunt. The infantile type of coarctation of the aorta will not be considered in this text.

The *adult type* is the form of coarctation which is usually encountered clinically. It consists of a more localized and less severe narrowing of the aorta at or just distal to the insertion of the ductus arteriosus. Postnatally, the ductus arteriosus is often closed; however, if the ductus remains patent after birth, the ductal shunt is directed from left to right—that is, from aorta to pulmonary artery.

Hemodynamically and prognostically, the principal difference between the infantile and adult types of coarctation of the aorta is that a fully developed collateral circulation is present at birth in the adult type of coarctation and is absent in the infantile type. The development of a collateral circulation in utero depends on whether or not, despite the coarctation, the fetal circulation is adequate. Apparently, in the infantile type of coarctation an adequate fetal circulation is maintained for either or both of the following reasons: (a) the ductus arteriosus inserts distal to the site of constriction of the aorta and therefore is able to shunt blood into the distal descending aorta; (b) the ductus arteriosus is malformed and greatly widened and therefore shunts a large enough volume

* Composite overloading of the right or left ventricle is characterized electrocardiographically by the presence of signs of both diastolic and systolic overloading.

of blood to satisfy the demands of the fetal circulation In either case in the infantile type of coarctation the stimulus is lacking for the development of a collateral circulation in utero On the other hand in the adult type of coarctation of the aorta the ductus arteriosus is unable to maintain an adequate fetal circulation, either because of its location proximal to the coarctation or because of its normal width Thus if the coarctation is severe a collateral circulation develops in utero in contrast with the situation in the infantile type

Typically in coarctation of the aorta there is hypertension in the upper extremities and weak or absent arterial pulsations in the lower extremities The

but two mechanisms have been entertained (a) some authorities attribute the hypertension to mechanical obstruction of blood flow by the coarctation itself while (b) others implicate renal ischemia secondary to the diminished blood flow to the kidneys There is evidence for and against both mechanisms In any event, depending on the degree of coarctation and the extent of the collateral circulation there is systolic overloading of the left ventricle whether this is attributable to the mechanical effects of the coarctation to hypertension of renal origin or to both factors In addition, the hemodynamic burden imposed on the left ventricle is sometimes increased even further by associated cardiac malformations the most common of which is a bicuspid aortic valve In the presence of the latter anomaly aortic insufficiency can result from incompetence of the aortic cusps due to hypertension and to dilatation of the aorta proximal to the coarctation.

In brief if there is marked stenosis of the aorta or if the collateral circulation is poorly developed coarctation of the aorta may produce severe systolic overloading of the left ventricle which in turn may cause concentric hypertrophy of the left ventricle On the other hand if the coarctation is of a relatively mild degree or the collateral circulation well developed, there may be minimal left ventricular hypertrophy Obviously all degrees of anatomic left ventricular hypertrophy between the preceding two extremes may be encountered

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lar lesions for example produce eccentric hypertrophy of the left ventricle and therefore exhibit electrocardiographic left axis deviation is that these lesions are often accompanied by myocardial disease and so there is both systolic and diastolic overloading of the left ventricle

4 Complete or incomplete right bundle branch block was present in about 30% of the cases of the combined series of Ziegler Metianu and his co workers and Kjellberg and his associates In the series of coarctation cases reported by Metianu and his collaborators right bundle branch block was observed in about 15% of the cases and left bundle branch block in about 25% However left bundle branch block was not reported to have occurred in any of the other series of cases reviewed by us

5 According to Landtman depressed S–T segments appeared in leads II and V_5 in about one fourth of the cases he studied but inverted T waves were never present in lead I and only exceptionally present in leads II and V_5 The relative paucity of T wave abnormalities in *left* ventricular hypertrophy due to coarctation is in rather striking contrast to their frequency in left ventricular hypertrophy due to other cardiac lesions and this fact does not seem consistent with Cabrera and Monroy s concept of the electrocardiographic changes attributable to systolic overloading of the left ventricle

Congenital Aortic and Subaortic Stenosis

Congenital aortic (valvular) stenosis is the result of fusion of the valve cusps while subaortic stenosis *is due to a localized constriction of the infundibulum* of the left ventricular outflow tract Depending on the degree of stenosis both lesions may fail to cause significant hypertrophy of the left ventricle or on the other hand they may cause severe systolic overloading of the left ventricle with resulting concentric ventricular hypertrophy

The ECG and VCG Findings

The findings of Braunwald and Kjellberg and their associates follow

1 In the combined series of cases reported by Braunwald and his co workers and Kjellberg and his associates approximately 85% of the cases showed normal electrocardiograms, while the remaining cases showed electrocardiographic left ventricular hypertrophy patterns

2 Braunwald and his collaborators observed left axis deviation in the electrocardiogram in only 1 of 12 cases of congenital aortic stenosis and subaortic stenosis

3 Braunwald and his associates studied their 12 pa [illegible] electro

say vectorcardiographic studies of this type of cardiac anomaly are relatively few in number

STENOTIC LESIONS CAUSING SYSTOLIC RIGHT VENTRICULAR OVERLOADING

Pulmonic Stenosis (Isolated with Normal Aortic Root)

Ordinarily pulmonic stenosis is not an isolated anomaly but clinically is accompanied by other congenital defects particularly septal defects and aortic overriding But even when pulmonic stenosis occurs *in company with other anomalies in most instances* it determines the severity of the hemodynamic burden imposed on the right ventricle While pulmonic stenosis due to narrowing of the valvular ring or to stenosis of the *pulmonary artery above the valve* (supravalvular) may be encountered on rare occasions the forms of pulmonic stenosis most frequently observed clinically fall into two main categories valvular and *infundibular stenosis*

Valvular stenosis involving the cusps of the pulmonary valve is the type said to predominate when pulmonic stenosis is associated with atrial septal defect or when it occurs in the absence both of aortic overriding and of defects of the atrial and ventricular septa

Infundibular stenosis stenosis of the infundibulum of the right ventricle may appear in either of the following two forms (*a*) a general narrowing of the *infundibulum from* its ostium up to the pulmonary valve which is found only with aortic overriding (see tetralogy of Fallot) (*b*) stenosis limited to the infundibular ostium the form most frequently associated *with ventricular* septal defects (without aortic overriding)

Valvular stenosis may exist separately or in combination with infundibular stenosis however infundibular stenosis unaccompanied by valvular pulmonic stenosis is said to be a rare finding although there is not universal agreement on this point

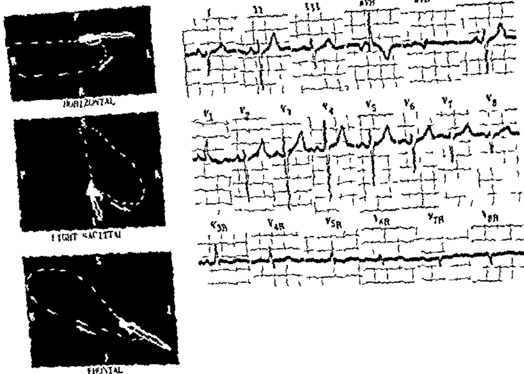


Fig 113 — Pl...

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Hemodynamically isolated pulmonic stenosis may be of such a minimal degree as to impose little or no additional burden on the right ventricle or, as is more often the case, it may greatly increase the resistance against which the right ventricle must eject blood during systole.

The resulting systolic overloading of the right ventricle can cause varying degrees of concentric right ventricular hypertrophy (without dilatation). Diastolic filling of the hypertrophied right ventricle requires increased pressure, and this is accompanied by a rise in pressure in the right atrium. Thus in rather severe isolated pulmonic stenosis there may occur both concentric hypertrophy of the right ventricle and hypertrophy and dilatation of the right atrium.

THE ECG AND VCG FINDINGS

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1. The electrocardiogram was normal in a little over 20% of the 28 cases.
2. The P waves in lead II of the electrocardiogram were abnormally tall and/or wide in 16% of the cases.
3. In almost two thirds of the cases the electrocardiogram was diagnostic of right ventricular hypertrophy, but the vectorcardiogram was diagnostic of right ventricular hypertrophy in all 25 of the cases studied. In 21 of the vectorcardiograms the QRS sE loop displayed the tall R type of right ventricular hypertrophy pattern, while the remaining 4 records showed RSR right ventricular hypertrophy patterns.
4. Right bundle branch block was present in about 10% of the electrocardiograms.
5. Almost 60% of the electrocardiograms showed right axis deviation; the remaining records were divided evenly in showing no axis deviation and left axis deviation.
6. Although some authorities have reported a parallel relationship between the level of right ven

tricular pressure in pulmonic stenosis and the prominence of the electrocardiographic and vectorcardiographic findings of right ventricular hypertrophy. Braunwald and his associates were unable to confirm this observation. However, the authors of this text have observed a general relationship between the severity of the clinical manifestations of pulmonic stenosis and the prominence of the electrocardiographic and vectorcardiographic abnormalities.

CARDIAC SHUNTS CAUSING DIASTOLIC LEFT VENTRICULAR OVERLOADING WITH OR WITHOUT SYSTOLIC RIGHT VENTRICULAR OVERLOADING

Interventricular Septal Defect

Unlike atrial septal defects, ventricular septal defects occur more frequently in association with other congenital cardiac anomalies than as isolated lesions. A common combination is ventricular septal defect and pulmonic (usually infundibular) stenosis. At least 90% of isolated ventricular septal defects and virtually all such lesions combined with other cardiac malformations represent developmental anomalies of the membranous portion of the interventricular septum. All other ventricular septal defects result from imperfect development of the muscular septum (maladie de Roger) and are usually so small as to be hemodynamically insignificant. The severity of the clinical manifestations of ventricular septal defect is related to the direction and magnitude of the interventricular shunt, these factors in turn being influenced by the size of the septal patency and the pressure gradient between the two ventricles. In the absence of associated anomalies, such as severe pulmonic stenosis or an overriding aorta, the pressure gradient usually produces a left to right shunt. However, changes in the peripheral vascular resistance of the pulmonary

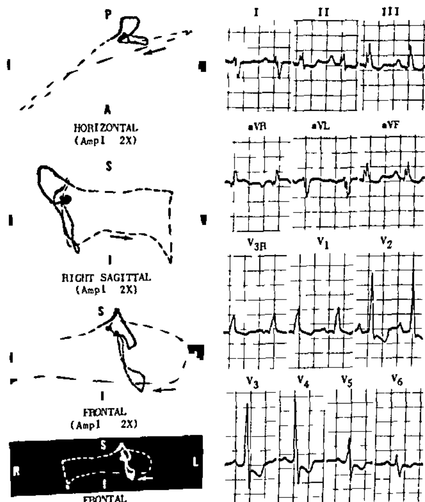
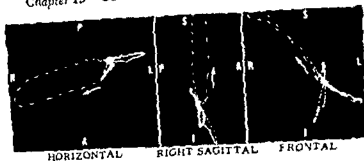


Fig. 114.—Electrocardiographic and vectorcardiographic tall R pattern of right ventricular hypertrophy in a woman, 28, with a large interventricular septal defect and pulmonary hypertension. The initial QRS forces, presumably due to septal ac...

...in leads I and V_6 of the electrocardiogram. The corresponding abnormal...

...slightly anteriorly. The large P sE loop is oriented slightly anteriorly, to the left, and inferiorly, and its terminus is displaced superiorly, to the right, and posteriorly. These findings are somewhat suggestive of right atrial enlargement (P pulmonale). Note also that the QRS sE loop remains open in each projection, indicating the presence of an S–T vector directed to the right and superiorly.



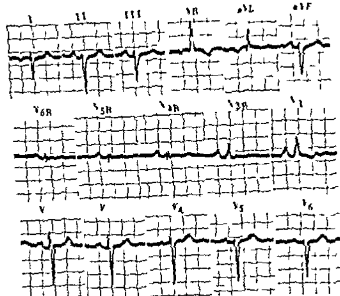


Fig 115 —Electrocardiographic and vectorcardiographic tall R pattern of right ventricular hypertrophy in a woman 30 with postmortem findings of cor triloculare biventriculare with common atrioventricular valve corrected transposition of the great vessels valvular pulmonic stenosis totally anomalous pulmonary venous drainage and situs inversus of liver stomach and pancreas Note the inverted P wave in lead I which correlates with the rightward orientation of the P sE loop of the vectorcardiogram

and systemic circulations also can alter the magnitude and direction of the shunt.

Sodi Pallares and his associates have found the following relationships to hold true in ventricular septal defect (a) If the right ventricular pressure is less than 60 mm Hg there is systolic overloading of the right ventricle and diastolic overloading of the left ventricle (b) If the right ventricular pressure exceeds 60 mm Hg there is more marked systolic overloading of the right ventricle but evidence for diastolic overloading of the left ventricle is lacking in the electrocardiogram

Kjellberg and his collaborators have pointed out that if in a ventricular septal defect the peripheral resistance in the pulmonary circulation equals that in the systemic circulation a significant interventricular shunt does not occur and so the hemodynamic burden of the left ventricle is not increased, while that of the right ventricle is greatly increased This leads to isolated right ventricular hypertrophy However with large interventricular septal defects there is a large interventricular shunt despite a markedly elevated pulmonary vascular resistance so that more often than not anatomic hypertrophy of both left and right ventricles occurs

The ECG and VCG Findings

The data presented below were derived from the combined series of cases reported by the following investigators Braunwald Hubbard Marisco and Kjellberg and their associates and includes 9 cases of the authors of this text The combined series which contains uncomplicated ventricular septal defects and septal defects with right to-left shunts totals 238 cases but only 25 of the patients were studied vectorcardiographically

1 The electrocardiogram was normal in 10% of all the cases In the smaller group of patients studied vectorcardiographically 40% of the vectorcardiograms were normal However this finding was confined entirely to those patients who had a

ventricular septal defect without complications

2 The P waves were abnormally tall and/or wide in leads I II or V_1 in almost 15% of the cases. Braunwald and his associates make no mention of the P sE loop findings in their reported series of congenital cardiac anomalies studied vectorcardiographically. None of the 5 patients with ventricular septal defect studied vectorcardiographically by us exhibited P sE loop findings unequivocally suggestive of atrial enlargement.

3 The diagnostic findings of left ventricular hypertrophy were present in 23% of the electrocardiograms and 12% of the vectorcardiograms.

4 The electrocardiogram was diagnostic of right ventricular hypertrophy in almost 35% of the cases while 45% of the patients studied vectorcardiographically showed right ventricular hypertrophy patterns primarily of the tall R type.

5 The electrocardiographic diagnosis of combined ventricular hypertrophy was made in 13% of the cases and complete or incomplete right bundle branch block was present in 7%.

6 About 10% of the cases showed first degree atrioventricular block (prolonged P–R interval) while over 15% exhibited the Katz Wachtel sign consisting of diphasic QRS complexes of great voltage in leads V_2 V_3 and V_4 as well as in the standard limb leads. Sodi Pallares believes the Katz Wachtel sign is relatively specific for ventricular septal defect although occasionally it is observed in atrial septal defect and in patent ductus arteriosus.

7 The mean manifest electrical axis of QRS was normal in 34% of the cases. Right axis deviation was present in 40% of the cases and left axis deviation in 26%.

8 Sodi Pallares and his co workers have observed deep Q waves in the left precordial leads of patients with ventricular septal defect and attribute this finding to septal hypertrophy. They regard deep Q waves in the left precordial leads as suggestive evidence favoring ventricular septal defect over other lesions (such as patent ductus arteriosus) which like the septal defect can produce combined ventricular hypertrophy.

In general the electrocardiographic and vectorcardiographic findings can be correlated with the clinical and hemodynamic features of a ventricular septal defect in the following way (*a*) A normal electrocardiogram and vectorcardiogram suggests that in the absence of pulmonic stenosis the right ventricular pressure is probably normal and either there is no interventricular shunt or there is a left to right shunt (*b*) If the electrocardiogram or vectorcardiogram shows isolated right ventricular hypertrophy or combined ventricular hypertrophy then it is very likely that pulmonary hypertension exists (Figs 114 and 115)

Patent Ductus Arteriosus

During fetal life the ductus arteriosus performs the necessary function of carrying blood from the pulmonary artery to the aorta. This short vessel originates near the bifurcation of the main pulmonary artery or from the left pulmonary artery and joins the aorta just distal to the insertion of the left subclavian artery. During the first postnatal year it is estimated that about 95% of ducti become obliterated although functional closure as opposed to anatomic obliteration probably occurs almost immediately following birth in normal infants. Carson and Burford feel that if the ductus arteriosus has not been obliterated by the time the child is 3 years old there is little likelihood of subsequent spontaneous closure. While asphyxia has been shown to delay closure of a normal ductus arteriosus persistent patency of the ductus is thought probably to reflect an actual malformation of the vessel and not simply a failure of the normal ductus to close. A patent ductus may occur as an isolated abnormality or in combination with other congenital anomalies such as septal defects pulmonic or aortic stenosis and malformations of the aortic arch system. Pulmonary hypertension may also complicate a patent ductus arteriosus and has been attributed to pulmonary vascular changes. However more recent observations lend some credence to the belief that in some instances at least pulmonary hypertension may be related to hypoxia and may disappear when the latter condition is corrected.

A patent ductus arteriosus produces an arteriovenous shunt between the aorta and the pulmonary artery. This shunt leads to an increased pulmonary circulation and an increased pulmonary artery pressure and concomitantly to a greater pulmonary venous return to the left heart. Thus during a given time interval the left ventricle may expel 2–4 times the volume of blood ejected by the right ventricle. For this reason an uncomplicated patent ductus arteriosus causes diastolic overloading of the left ventricle. If pulmonary hypertension supervenes in the course of a patent ductus then depending on the duration and severity of the resulting systolic overloading of the right ventricle the electrocardiographic and vectorcardiographic findings of combined ventricular hypertrophy or occasionally the findings of isolated right ventricular hypertrophy may appear

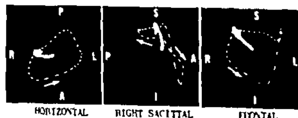
THE ECG AND VCG FINDINGS

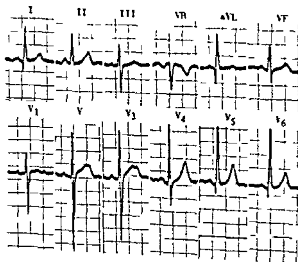
The material presented below is based on the combined series of Braunwald and his co-workers, Sokolow and Edgar, Kjellberg and his associates, and the authors of this text. The total number of cases in the groups, which includes cases of uncomplicated patent ductus arteriosus and cases with pulmonary hypertension, was 139; 17 of the patients were studied vectorcardiographically.

... the presence of combined ventricular hypertrophy might be suspected from the electrocardiogram, and this interpretation would be consistent with patent ductus arteriosus with early and relatively minor degree of pulmonary hypertension.

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1. The electrocardiogram was normal in about 60% of the cases, while approximately 45% of the vectorcardiograms were normal.
2. Almost one fourth of the cases presented electrocardiographic evidence of left ventricular hypertrophy, while 2 of the 17 vectorcardiograms were interpreted as left ventricular hypertrophy.
3. Eight per cent of the electrocardiograms showed right ventricular hypertrophy, and almost one third of the vectorcardiograms presented right ventricular hypertrophy.
4. ... ctorcardiograms recorded by us in patients with patent ductus arteriosus were unusual in that the QRS sÊ loops were situated more anteriorly than normal, although inscribed in a normal direction in the horizontal projection. In addition, the QRS sÊ loops showed a prominent early deflection extending relatively far to the right and anteriorly. These early deflections were undoubtedly responsible for the tall R waves recorded in lead V_1 in the electrocardiograms of these 2 cases. In each case, the R wave exceeded 7 mm, even though the R/S amplitude ratio in V_1 was less than 1. Parenthetically, one might add that in the 2 cases in question the patients were adults. As will be recalled, the electrocardiogram of the juvenile typically displays much taller R waves in lead V_1 than does that of the adult. The authors consider the electrocardiographic and vectorcardiographic findings just described, when observed in an adult, to be strongly suggestive of combined ventricular hypertrophy.
5. Incomplete or complete right bundle branch block was noted in the electrocardiograms of only 3% of the cases.
6. The mean manifest electrical axis of QRS was normal in 60% of the cases (Fig. 116); right axis deviation was present in 30% of the electrocardiograms, and left axis deviation was present in 10% of the tracings.





CARDIAC SHUNTS CAUSING DIASTOLIC RIGHT VENTRICULAR OVERLOADING

Interatrial Septal Defect (with Left to Right Shunt)

When there is a congenital defect in the interatrial septum (other than a patent foramen ovale) the pressure gradient normally existing between the left and *the right atria produces a left to right shunt of blood* through the septal defect Depending on the magnitude of the left to right shunt the right ventricular output can rise to twice that of the left ventricle and pulmonary blood flow may be increased to 3 times systemic blood flow An atrial septal defect therefore leads to diastolic overloading of the right ventricle According to Walker and his collaborators the muscles of the basal portion of the right ventricle bear for the most part the hemodynamic burden imposed by an increase in stroke volume consequent to diastolic overloading of the right ventricle *This is to be* contrasted with the concentric hypertrophy of the right ventricular wall which occurs in conditions increasing ventricular work—that is in conditions causing systolic overloading Thus Walker and his associates postulate that a selective hypertrophy of basal right ventricular muscle occurs in atrial septal defects In support of this hypothesis Kjellberg and his co-workers have reported that on pathologic examination the right ventricular wall is of normal thickness in cases of atrial septal defect without pulmonary hypertension *however characteristically there is marked* hypertrophy of the crista supraventricularis its parietal and septal bands and the trabecular network of the right ventricle With the development of pulmonary hypertension (and the resulting systolic overloading of the right ventricle) in some cases of atrial septal defect the right ventricle undergoes concentric hypertrophy The significance of these observations with reference to the electrocardiographic and vectorcardiographic features of atrial septal defect will be indicated later

Because in an atri*al septal defect the right atrium* receives blood from the venae cavae as well as from the left atrium the right atrium usually undergoes a variable degree of dilatation and hypertrophy On the other hand the left atrium is usually not enlarged even with large left to right shunts such as those caused by an associated mitral stenosis (Lutembacher's syndrome) since the atrial septal defect functions somewhat like a safety valve preventing any significant increase in the amount of blood in the left atrium

THE ECG AND VCG FINDINGS

The percentages of cases of atrial septal defect showing each of the electrocardiographic and vectorcardiographic features cited below were calculated from the combined series of the following investigators Braunwald Kjellberg Limon Silverblatt and Walker and their associates Smull and Lamb and the authors of this text When these series were pooled the number of cases totaled 239 but only 63 patients were studied vectorcardiographically

1 *The electrocardiogram was normal in about 6% of* the 239 patients while the vectorcardiogram was normal in none of the 63 patients studied vectorcardiographically
2 Abnormally tall and/or wide P waves were recorded in lead II and/or lead V_1 in about one fourth of the cases In our experience abnormalities of the P sÊ loop were relatively uncommon in vectorcardiograms recorded in cases of atrial septal defect
3 *The electrocardiogram was diagnostic of right ventricular hypertrophy in about 30% of the cases* while the vectorcardiogram presented findings diagnostic of right ventricular hypertrophy in about 95% of the cases so studied Approximately 60% of the vectorcardiograms showing right ventricular hypertrophy were found to have the RSR pattern [illegible]

vectorcardiographic pattern of right ventricular hypertrophy are atrial septal defect and mitral stenosis

4 A right bundle branch block type of QRS configuration with a QRS duration of 0 10 second or longer was present in lead V_1 in about 13% of the cases while RSR deflections of shorter duration in lead V_1 were found in about 55% of the cases *Although QRS sÊ loops otherwise typical of right* ventricular hypertrophy can occasionally display conduction delay localized to the terminal portion of the loop or involving the greater portion of the loop the vectorcardiographic pattern of right bundle branch block unaccompanied by evidence of right ventricular hypertrophy was encountered relatively rarely Thus Braunwald and his associates observed the vectorcardiographic pattern of right bundle branch block in only 1 of 19 cases of interatrial septal defect and we recorded this pat

tern in only 1 of 14 cases of interatrial septal defect

5 ... s deviation ... axis deviation ... axis deviation
tion in about ... tion in about 32%

6 According to Bellet, in interatrial septal defect there may be encroachment on or disease of the atrioventricular node The resultant P-R interval prolongation is said to increase the susceptibility to the development of atrial fibrillation Additional factors which may be operative are the increased work of the right heart the enlargement of the right atrium the presence of anoxia and the frequent association of congestive heart failure However ... the ... ators

and apparently these 3 cases with atrial arrhythmias were instances of Lutembacher's syndrome

Braunwald and his co-workers make no mention of finding atrial flutter or fibrillation in any of these patients with interatrial septal defect and we observed atrial fibrillation in only 1 of our 15 patients

7 Complete atrioventricular block may on rare occasions accompany an interatrial septal defect *but* prolonged atrioventricular conduction (first-degree atrioventricular block) is a more common finding Limon and his collaborators observing this *abnormality in* about 25% of their cases

Atrial Septal Defect (with Right-to-Left Shunt)

Trilogy of Fallot (pulmonic stenosis with interatrial septal defect or patent foramen ovale)—This combination of cardiac anomalies clinically resembles tetralogy of Fallot As a general rule the hemodynamic burden placed on the right *ventricle* exceeds that imposed by an isolated atrial septal *defect* and is de

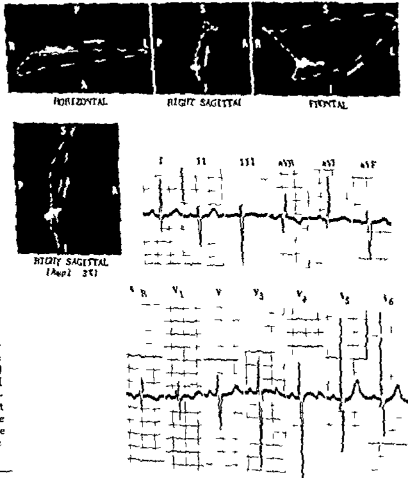


Fig 117 —Electrocardiographic and vectorcardiographic RSR' pattern of right ventricular hypertrophy in a boy 16 The patient expired after cardiac surgery and at *postmortem* was found to have a patent foramen ovale an interatrial septal defect hypertrophy and dilatation of the right ventricle and situs *inversus* of the abdominal viscera. While the right precordial leads of the electrocardiogram display RSR' deflection of 0 08-second duration suggesting right ventricular hypertrophy the left axis deviation of Â QRS and the tall R waves in leads V and V on the other hand, are suggestive of left ventricular hypertrophy In the vectorcardiogram the horizontal QRS loop pattern is that of the RSR pattern of right ventricular hypertrophy However the superior orientation of the sagittal and frontal QRS loops and the counterclockwise direction of inscription of the latter loop are unusual findings on this vectorcardiographic pattern of right ventricular hypertrophy While some authorities consider the features just described to be indicative of coexisting left and right ventricular hypertrophy the observations of the authors of this text are in conflict with this opinion We believe the left axis deviation to be suggestive of a septum primum defect

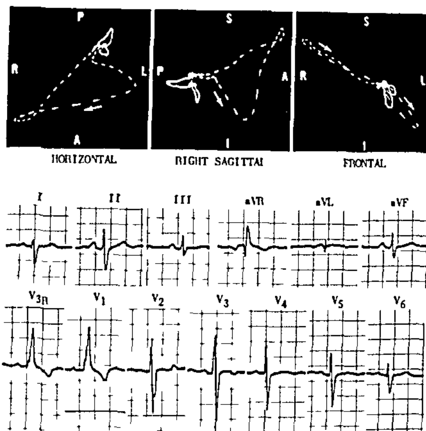


Fig 118 —Electrocardiographic and vectorcardiographic tall R pattern of right ventricular hypertrophy in a youth 19 with pulmonic stenosis and interatrial septal defect (trilogy of Fallot)

termined by the degree of pulmonic stenosis Generally this condition is also accompanied by more prominent electrocardiographic and vectorcardiographic findings of right ventricular hypertrophy than occur in uncomplicated atrial septal defects (Fig 118)

Atrial septal defect with pulmonary hypertension —A reversed or right to left shunt produced by pulmonary hypertension is a much less common occurrence with atrial septal defect than with ventricular septal defect or patent ductus arteriosus As in trilogy of Fallot the degree of anatomic right ventricular [illegible]

1

septal defect with pulmonary hypertension on the basis of pulmonary vascular disease than in uncomplicated septal defects

MULTIPLE CARDIAC ANOMALIES OR CARDIAC ANOMALIES ACCOMPANIED BY OTHER DEVELOPMENTAL DEFECTS

Dextrocardia

When the heart is located in the right chest any of the following abnormalities may be responsible

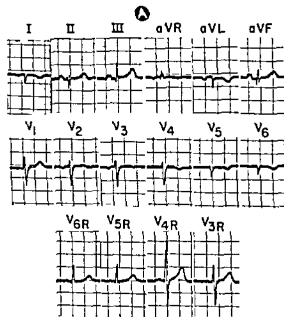
Dextrocardia with situs inversus —In this condition there is true or mirror image dextrocardia in which the left sided chambers are situated to the right while the right sided heart chambers are located to the left The apex of the heart is formed by the left ventricle This is the type of dextrocardia which with chronic sinusitis and bronchiectasis comprises Kartagener s triad (or syndrome)

Isolated dextrocardia —In this instance mirror image or true dextrocardia is not accompanied by situs inversus of other body organs but tends to be associated with other cardiac anomalies such as pulmonic stenosis or cor triloculare biatriatum

Dextroversion of the heart —Mirror image dextrocardia is not present in this condition The heart is merely displaced and rotated so that it lies in the right chest and there are usually associated cardiac abnormalities

Dextroposition of the heart —In this condition the heart is merely displaced into the right chest by some acquired disease of the lungs pleura or diaphragm

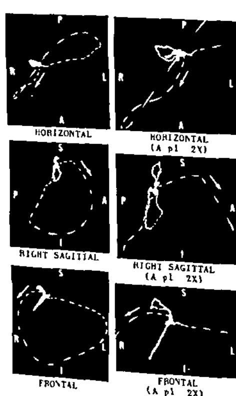
Fig 119 —A electrocardiographic features of [illegible]

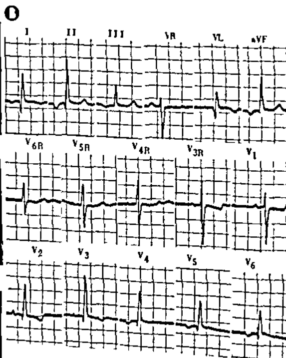


and the precordial leads were recorded from left

[illegible]

[illegible] hypertrophy in a woman [illegible] with Eisenmenger's complex (large interventricular septal defect with overriding aorta and pulmonary hypertension) and total situs inversus Here the electrocardiographic findings differ from those in [illegible]





The ECG and VCG Findings

In the following paragraphs the electrocardiographic and vectorcardiographic findings in mirror image or true dextrocardia will be described The electrocardiographic features of dextrocardia are as follows

1 The electrical axis of P (Â P) is situated at about +120° in the frontal plane with the result that *lead I records an inverted P wave* This is the most typical finding in dextrocardia
2 The QRS deflection in lead I and the T wave in this lead are *mirror images of the corresponding* normal deflections in this lead Thus there is usually a deep Q wave and an inverted T wave in lead I
3 Leads II and III are interchanged
4 The precordial transition is reversed so that leads over the right precordium record upright QRS deflections and upright T waves while leads over the left precordium record resultantly downwardly directed ventricular deflections
5 If the left and right arm lead wires are reversed and the precordial leads are recorded from left to right over the right precordium the electrocardiogram recorded should appear normal If it does not then associated cardiac abnormalities should be suspected (Fig 119 A)

The vectorcardiographic findings in mirror image or true dextrocardia are as follows

1 The P sÊ loop is usually directed to the right inferiorly and either slightly anteriorly or slightly posteriorly The direction of the inscription of the P sÊ loop in the right sagittal projection is the reverse of normal i e counterclockwise
2 The QRS sÊ loop is generally situated to the right inferiorly and slightly anteriorly or slightly posteriorly The direction of inscription of the spatial loop in each of its three projections is the reverse of normal (Fig 119 B)

Tetralogy of Fallot

The familiar tetrad of abnormalities collectively designated *tetralogy of Fallot* consists of three congenital anomalies—namely pulmonic stenosis aortic overriding and high interventricular septal defect—*which jointly* produce the remaining acquired abnormality right ventricular hypertrophy This condition accounts for about 75% of all cases of cyanotic congenital heart disease Fortunately certain surgical procedures achieve remarkably beneficial results in many patients with tetralogy of Fallot

The basic hemodynamic disturbance produced by this combination of anomalies is a right to left shunt between the right ventricle and the aorta Whether the clinical consequences of a tetralogy *are minimal* lethal or of some intermediate degree of severity is determined mainly by the magnitude of the shunt or even more fundamentally by the degree of pulmonic *stenosis and overriding of the aorta Thus the pul*monic stenosis may be mild the aortic overriding minimal and the hemodynamic disturbance relatively insignificant The other extreme is represented by pulmonary atresia and complete transposition of the aorta the latter arising entirely from the right ventricle This combination is sometimes called *pseudo truncus arteriosus* In the less extreme cases of tetralogy of Fallot there may be both systolic and diastolic overloading of the right ventricle according to Cabrera and Monroy These investigators state that the pulmonary stenosis and the overriding of the aorta determine the systolic overloading while the blood shunted from the right ventricle to the aorta increases the diastolic loading of the right cavities From the standpoint of the electrocardiogram Sodi Pallares and Marisco have found tetralogy of Fallot to behave much like a pure pulmonic stenosis

The ECG and VCG Findings

The percentages of cases of tetralogy of Fallot showing the various findings cited below were calculated from the combined series of Braunwald and his co workers Kjellberg and his associates Woods and the authors of this text The number of cases in the combined series totaled 125 of which 30 were studied vectorcardiographically

1 Both the electrocardiogram and vectorcardiogram were normal in about 3% of the cases
2 In about 30% of the cases the P waves in lead II and/or lead V_1 were abnormally tall and/or wide
3 In approximately 95% of the cases the electrocardiogram and vectorcardiogram presented the findings of right ventricular hypertrophy the vectorcardiographic right ventricular hypertrophy pattern usually being of the tall R type
4 The electrocardiographic pattern of complete or incomplete right bundle branch block was observed in about 3% of the cases
5 There was right axis deviation of the mean manifest electrical axis of QRS in almost 90% of the cases left axis deviation in about 7% and no axis deviation in the remaining 3% of the cases

Mitral Stenosis

ORDINARILY in a discussion of the pathophysiologic factors responsible for the electrocardiographic and vectorcardiographic findings in mitral stenosis chief and almost exclusive attention is given to the mechanical effects of the stenotic mitral valve and the associated rise in pulmonary capillary and arteriolar pressures However it should not be forgotten that invariably in rheumatic valvular lesions such as mitral stenosis antecedent rheumatic carditis has left residual myocardial damage of varying degree Sometimes the myocardial damage may actually assume greater importance than the valvular lesion and may determine among other things the clinical course and the electrocardiographic manifestations of the patient s cardiac disease Since the significance of the myocardial factor in a given case of mitral stenosis is difficult and usually impossible to assess before autopsy the hemodynamic and related electrical effects of mitral stenosis will be considered in the following paragraphs only from the standpoint of the valvular lesion itself and the accompanying changes *in the* pulmonary circulation

The train of pathophysiologic events in mitral stenosis can be described in brief as follows

1 The increased resistance offered by *the stenotic* mitral valve leads to an elevated pressure in the left atrium and since there are *no* valves between the atrium and the pulmonary veins the increased left intra atrial pressure induces a rise in the pulmonary venous and capillary pressures

2 Any increase in blood flow such as that occurring during exercise increases left atrial and pulmonary capillary pressures Moreover the stenotic mitral valve sometimes offers relatively little resistance to blood flow at rest but with exercise its ability to adjust to the greater blood flow is limited and this can be reflected by a marked rise in left atrial pressure

3 The pulmonary arterial pressure for a given blood flow is a function of the pulmonary capillary pressure (measured as the so-called *wedge pressure*) and the resistance in the pulmonary arterioles If an elevation of the pulmonary capillary pressure is of sufficiently long duration pulmonary vascular changes develop in which case the resulting increased arteriolar resistance in conjunction with the elevated pulmonary capillary pressure produces pulmonary hypertension

4 To overcome the greater resistance to systolic ejection consequent to the increased pulmonary venous and capillary pressures and thereby to maintain its normal output the right ventricle must contract more forcefully This in turn leads to a rise in pulmonary arterial and right ventricular systolic pressures Concomitant elevation of the pulmonary arteriolar resistance has two contrasting hemodynamic consequences (*a*) the higher pulmonary arteriolar resistance on the *one hand* protects the pulmonary capillaries from marked rises in pressure which otherwise might produce pulmonary edema and (*b*) on the other hand the greater arteriolar resistance imposes a more severe hemodynamic burden on the right ventricle

In the presence of pulmonary hypertension there is systolic overloading *of the* right ventricle and if cardiac failure *supervenes* there may be added to this *hemodynamic* burden diastolic overloading of the right ventricle In such cases one not infrequently finds anatomic and electrocardiographic evidence of right ventricular hypertrophy of a degree comparable to that observed in some of the more severe types of congenital heart disease On the other hand the prominence of the electrocardiographic manifestations may fail to parallel the degree of the anatomically noted right ventricular hypertrophy because of the factors listed at the beginning of this section Two

of the more important of these variables in mitral stenosis are (*a*) changes in anatomic heart position and rotation which modify transmission of QRS potentials to the recording electrode and (*b*) the presence of additional valvular or myocardial lesions causing left ventricular hypertrophy (which tends to offset the electrical effects of the right ventricular hypertrophy)

In a typical case of mitral stenosis the right ventricle at postmortem examination shows little or no thickening of its muscular wall instead there is hypertrophy of the crista supraventricularis and related structures and of the trabeculae of the right ventricle This more or less selective hypertrophy of the right ventricle is usually accompanied by marked dilatation of the affected chamber In addition as one might anticipate mitral stenosis characteristically leads to dilatation and hypertrophy of the left atrium and sometimes of the right atrium The left ventricle may be relatively unaffected pathologically

ELECTROCARDIOGRAPHIC FINDINGS

The electrocardiographic findings most typical of mitral stenosis which are to be described in the following paragraphs are observed for the most part in *pure mitral stenosis* or in hemodynamically predominant mitral stenosis with coexisting mitral insufficiency of relatively minor significance With more severe degrees of mitral insufficiency or when mitral stenosis is associated with aortic valvular disease the electrocardiogram rarely displays the diagnostic findings of mitral stenosis

Typically in mitral stenosis the electrocardiographic manifestations are related etiologically to the two major anatomic effects of the valvular lesion which are as previously indicated left atrial enlargement and right ventricular hypertrophy To afford some estimate of the frequency of the various diagnostic features of pure or predominant mitral stenosis the series of cases of mitral stenosis of Lewis Bjorck Scott and Donoso and their associates and of Morris and Whitaker and Trounce including 47 cases of the authors of this text were combined to form a study group totaling 135 cases The percentage of electrocardiograms in this group showing each of the diagnostic features of mitral stenosis to be described will be cited at appropriate points in the following discussion

Anatomic left atrial enlargement per se can alter the electrocardiogram in two ways it can cause disturbances of the atrial rhythm and it can produce the P wave pattern of P mitrale

Cardiac Rhythm

Left atrial enlargement due to mitral stenosis frequently gives rise to paroxysmal or chronic atrial fibrillation or occasionally flutter The manner in which these atrial arrhythmias are produced in mitral stenosis is not known for certain although factors such as the following may be involved (*a*) elevated left atrial pressure (*b*) left atrial dilatation (*c*) intra atrial conduction delay consequent to atrial dilatation and/or to residual myocardial fibrosis following acute rheumatic carditis and (*d*) focal or diffuse fibrosis of atrial myocardium or acute "smoldering subacute rheumatic carditis

In the combined series of cases of mitral stenosis 20% of the electrocardiograms showed atrial fibrillation while 20% of the remaining cases with normal sinus rhythm in our own series displayed first degree atrioventricular block The latter finding is frequently noted electrocardiographically in patients with mitral stenosis before onset of atrial fibrillation and may persist indefinitely following conversion of atrial fibrillation to sinus rhythm Apparently in pure mitral insufficiency unaccompanied by mitral stenosis sinus rhythm is far more common than atrial fibrillation but in the great majority of cases of mitral stenosis with a significant degree of mitral insufficiency there is a very high incidence of atrial fibrillation (15 out of 17 cases reported by Wierum and Glenn)

The ECG and VCG P Mitrale Pattern

The abnormalities of the electrocardiographic P waves characteristic of the P mitrale pattern (Fig 120) are as follows

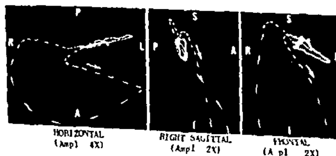
1 The mean manifest electrical axis of P (A P) or frontal plane mean P vector tends to assume a horizontal and leftward orientation approximating the 0 axis of the frontal reference frame in contrast with its normal average orientation along with +60 axis This means that by and large the P waves in leads I and aVL in P mitrale are more prominent than those in leads III and aVF
2 The P waves in leads I and aVL are abnormally wide exceeding the normal upper limit of P

wave duration of 0 11 second More often than not the P waves particularly in the leads just cited display double peaked flattened summits The notching and broadening of the P waves in P mitrale have been attributed by some au thorities to an intra atrial conduction disturb ance or to a prolonged activation time of the left atrium The conduction defect has been variously ascribed to stretching of the atrial wall as the left atrium dilates to interstitial fibrosis and to myocarditis Sano and his asso ciates believe that the characteristic widening and notching of the P waves in P mitrale simply reflect an exaggeration of the normal time lag of left atrial activation behind right atrial acti vation Presumably hypertrophy and dilatation of the left atrium increase the time required for activation and since the onset of activation in the left atrium normally occurs later than in the right atrium the result is an over all lengthening of the time required for depolar ization of both atria Moreover since activation of the right atrium tends to be separated in time from activation of the left atrium the electrical forces produced by activation of each atrium are manifested more or less separately in the electrocardiogram as evidenced by the double peaked P wave typical of P mitrale

Occasionally in mitral stenosis the P waves in leads I and aVL or less commonly in leads II and III are abnormally tall equaling or exceed ing 2 5 mm in amplitude

3 The mean instantaneous P wave forces and the horizontal plane mean P vector in mitral ste nosis tend to be rotated to the left and far pos teriorly probably reflecting the increased elec trical predominance of the posteriorly situated left atrium Alternatively the early instantane ous P forces which are attributed to right atrial activation are directed anteriorly but the sub sequent and larger electrical P forces produced by left atrial activation are displaced abnor mally far posteriorly Thus the P waves in leads V_1 and V may be large and downwardly di





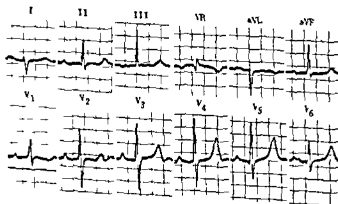


Fig 120 —Electrocardiographic and vectorcardiographic P mitrale pattern of left atrial enlargement and tall R pattern of right ventricular hypertrophy in a wom n 31 ...

cated at about 0 in the frontal refer ence frame while the width of the P wave in lead I is 0 13 second (upper limit of normal P wave duration 0 11 second) The P sE loop in the vector cardiogram is oriented almost directly to the left in the frontal projection and somewhat posteriorly in the horizontal projection The vectorcardiographic findings characteristic of the tall R pattern of right ventricular hypertrophy are obvious in this figure and need not be described

of the more important of these variables in mitral stenosis are (*a*) changes in anatomic heart position and rotation which modify transmission of QRS potentials to the recording electrode and (*b*) the presence of additional valvular or myocardial lesions causing left ventricular hypertrophy (which tends to offset the electrical effects of the right ventricular hypertrophy)

In a typical case of mitral stenosis the right ventricle at postmortem examination shows little or no thickening of its muscular wall instead there is hypertrophy of the crista supraventricularis and related structures and of the trabeculae of the right ventricle This more or less selective hypertrophy of the right ventricle is usually accompanied by marked dilatation of the affected chamber In addition as one might anticipate mitral stenosis characteristically leads to dilatation and hypertrophy of the left atrium and sometimes of the right atrium The left ventricle may be relatively unaffected pathologically

ELECTROCARDIOGRAPHIC FINDINGS

The electrocardiographic findings most typical of mitral stenosis which are to be described in the following paragraphs are observed for the most part in *pure mitral stenosis* or in hemodynamically predominant mitral stenosis with coexisting mitral insufficiency of relatively minor significance With more severe degrees of mitral insufficiency or when mitral stenosis is associated with aortic valvular disease the electrocardiogram rarely displays the diagnostic findings of mitral stenosis

Typically in mitral stenosis the electrocardiographic manifestations are related etiologically to the two major anatomic effects of the valvular lesion which are as previously indicated left atrial enlargement and right ventricular hypertrophy To afford some estimate of the frequency of the various diagnostic features of pure or predominant mitral stenosis the series of cases of mitral stenosis of Lewis Biorck Scott and Donoso and their associates and of Morris and Whitaker and Trounce including 47 cases of the authors of this text were combined to form a study group totaling 435 cases The percentage of electrocardiograms in this group showing each of the diagnostic features of mitral stenosis to be described will be cited at appropriate points in the following discussion

Anatomic left atrial enlargement per se can alter the electrocardiogram in two ways it can cause disturbances of the atrial rhythm and it can produce the P wave pattern of P mitrale

Cardiac Rhythm

Left atrial enlargement due to mitral stenosis frequently gives rise to paroxysmal or chronic atrial fibrillation or occasionally flutter The manner in which these atrial arrhythmias are produced in mitral stenosis is not known for certain although factors such as the following may be involved (*a*) elevated left atrial pressure (*b*) left atrial dilatation (*c*) intra atrial conduction delay consequent to atrial dilatation and/or to residual myocardial fibrosis following acute rheumatic carditis and (*d*) focal or diffuse fibrosis of atrial myocardium or acute "smoldering" subacute rheumatic carditis

In the combined series of cases of mitral stenosis 20% of the electrocardiograms showed atrial fibrillation while 20% of the remaining cases with normal sinus rhythm in our own series displayed first degree atrioventricular block The latter finding is frequently noted electrocardiographically in patients with mitral stenosis before onset of atrial fibrillation and may persist indefinitely following conversion of atrial fibrillation to sinus rhythm Apparently in pure mitral insufficiency unaccompanied by mitral stenosis sinus rhythm is far more common than atrial fibrillation but in the great majority of cases of mitral stenosis with a significant degree of mitral insufficiency there is a very high incidence of atrial fibrillation (15 out of 17 cases reported by Wierum and Glenn)

The ECG and VCG P Mitrale Pattern

The abnormalities of the electrocardiographic P waves characteristic of the P mitrale pattern (Fig 120) are as follows

1 The mean manifest electrical axis of P (A P) or frontal plane mean P vector tends to assume a horizontal and leftward orientation approximating the 0 axis of the frontal reference frame in contrast with its normal average orientation along with +60 axis This means that by and large the P waves in leads I and aVL in P mitrale are more prominent than those in leads III and aVF
2 The P waves in leads I and aVL are abnormally wide exceeding the normal upper limit of P

wave duration of 0 11 second More often than not the P waves particularly in the leads just cited display double peaked flattened summits The notching and broadening of the P waves in P mitrale have been attributed by some authorities to an intra atrial conduction disturbance or to a prolonged activation time of the left atrium The conduction defect has been variously ascribed to stretching of the atrial wall as the left atrium dilates to interstitial fibrosis and to myocarditis Sano and his associates believe that the characteristic widening and notching of the P waves in P mitrale simply reflect an exaggeration of the normal time lag of left atrial activation behind right atrial activation *Presumably* hypertrophy and dilatation of the left atrium increase the time required for activation and since the onset of activation in the left atrium normally occurs *later than in* the right atrium the result is an over all lengthening of the time required for depolarization of both atria. Moreover since activation of the right atrium tends to be separated in time from activation of the left atrium the electrical forces produced by activation of each atrium are manifested more or less separately in the electrocardiogram as evidenced by the double peaked P wave typical of P mitrale

Occasionally in mitral stenosis the P waves in leads I and aVL, or less commonly in leads II and III are abnormally tall equaling or exceeding 2 5 mm in amplitude

3 The mean instantaneous P wave *forces* and the horizontal plane mean P vector in mitral stenosis tend to be rotated to the left and far posteriorly probably reflecting the increased electrical predominance of the posteriorly *situated* left atrium Alternatively the early instantaneous P forces which are attributed to right atrial activation are directed anteriorly but the subsequent and larger electrical P *forces produced* by *left atrial activation* are displaced abnormally *far* posteriorly Thus the P waves in leads V_1 and V may be large and downwardly di

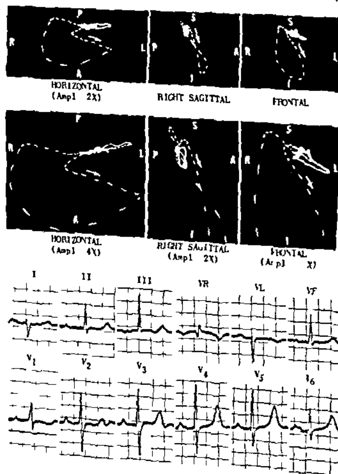


Fig 120 —Electrocardiographic and vectorcardiographic P mitrale pattern of left atrial enlargement and tall R pattern of right ventricular hypertrophy in a woman 31 with mitral stenosis The electrocardiographic diagnosis of left atrial enlargement is based on the following findings A I is located at about 0 in the frontal reference frame while the width of the I wave in lead I is 0 13 second (upper limit of normal I wave duration 0 11 second) The I sE loop in the vectorcardiogram is oriented almost directly to the left in the frontal projection and somewhat posteriorly in the horizontal projection The vectorcardiographic findings characteristic of the tall R pattern of right ventricular hypertrophy are obvious *in this* figure and need not be described

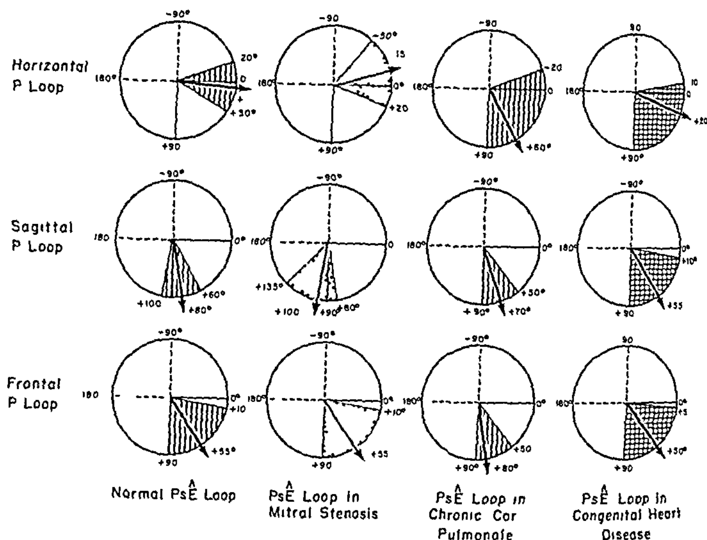


Fig 121 —Extreme range of variation and average orientation of the maximal mean instantaneous vector of the P sÊ loop in normal subjects in mitral stenosis in chronic cor pulmonale and in congenital heart disease

rected, on the one hand or they may be diphasic (plus minus)

In the mitral stenosis cases with sinus rhythm in *the combined series of Lewis and his co-workers* Scott and his associates and the authors of this text almost 65% of the electrocardiograms displayed abnormally wide and or tall P waves in one or more leads In our 30 cases with sinus rhythm the P waves were normal in 9 cases they were *abnormally wide in* lead I in 5 cases and in lead II in 9 cases 4 electrocardiograms displayed abnormally tall P waves in lead II and in 2 cases the P waves in lead II were both wide and tall In 1 case the P waves were abnormally wide in lead I and abnormally tall in lead II Notching of the P waves of varying degree was present in all 21 cases with other P wave abnormalities The P *wave amplitude in lead* V_1 *was not measured in our* cases of mitral stenosis but as a general rule the P waves in this lead were usually relatively large and either inverted or diphasic (plus minus) the initial upright component of the diphasic P wave being smaller than the following downwardly directed component The mean manifest electrical axis of P in the electrocardiograms with P wave abnormalities averaged +48 with a range of variation between 0° and +80 *It is worthy of note that left atrial enlargement* resulting from mitral insufficiency is associated only infrequently with P wave abnormalities Even when abnormal P waves are observed in mitral insufficiency they tend to be less striking than those occurring in *mitral stenosis* Brigden and Leatham studied 30 patients with relatively pure mitral insufficiency and found that the P waves were normal in all cases Thus when the electrocardiogram shows prominent P wave abnormalities the presence of mitral stenosis should be suspected

The vectorcardiographic P sÊ loop abnormalities

TABLE 12 —ORIENTATION OF THE MAXIMAL MEAN INSTANTANEOUS VECTOR OF THE P sÊ LOOP IN MITRAL STENOSIS

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	A	Usual Range	Extreme Range	A	Usual Range	Extreme Range	A	Usual Range
30 cases with mitral stenosis and sinus rhythm	−50 to +20	−15	30 to 0	+80 to +135	+100	+80 to +100	+10 to +90	+55	+30 to +80
16 (of the above 30) cases with electrocardiographic P mitrale	−50 to 0	−25	−30 to 0	+80 to 130	+105	+80 to +100	+10 to +80	+45	+30 to +70
(Normal cases)	(−20 to +30)	(+5)	(0 to +5)	(+60 to +100)	(+60)	(+80 to +90)	(+10 to +90)	(+55)	(+20 to +60)

The normal values for P sÊ loop orientation are enclosed in parentheses and are the same as those presented in Table 4 in Chapter 7. They are included in the above table to provide a basis for comparison with those obtained in mitral stenosis

corresponding to the electrocardiographic P mitrale pattern (Fig 121) are as follows

1 The maximal mean instantaneous P vector or long axis of the P sÊ loop is of greater than normal magnitude
2 In the horizontal projection the P sÊ loop in addition to its increased size frequently has a bifid or figure-of-eight configuration with the larger component of the loop being directed posteriorly and inscribed in a clockwise direction The earlier portion of the P sÊ loop is written anteriorly in a counterclockwise direction If the anterior component of the horizontal P loop equals or approaches in size the later posterior component both left and right atrial enlargement may be present The average orientation and range of variation of the P sÊ loop in our 30 cases of mitral stenosis are presented in Table 12 and in Figure 121 The average orientation of the P sÊ loop and range of variation were also determined in the 16 mitral stenosis cases with electrocardiographic P *mitrale* and these results are also indicated in Table 12
3 In the sagittal and frontal projections the P sÊ loop *is* elongated and may have a figure-of-eight configuration although far less frequently than in the horizontal projection The direction of inscription of the sagittal and frontal loops is the same as normal—that is clockwise and counterclockwise respectively

Right Ventricular Hypertrophy

If any one lead can be singled out as the diagnostic lead in right ventricular hypertrophy *occurring in mitral stenosis* it is lead V_1 because more often than not the electrocardiographic manifestations of right ventricular hypertrophy are most prominent and *most easily recognized* in this lead The combined series of cases of mitral stenosis were grouped according to the type of QRS configuration and/or the R/S amplitude ratio in lead V_1 and the following observations were made

1 Lead V_1 recorded an rS or QS deflection in *almost* 45% of the cases even though anatomic right ventricular hypertrophy could be demonstrated radiologically *in* many of these cases
2 In approximately 25% of the cases lead V_1 registered an RSR deflection The terminal R wave was of equal *or smaller* size than the initial R wave *in 10%* of the electrocardiograms while in the remaining 15% the secondary R wave was taller than the *initial* R wave The first type of RSR

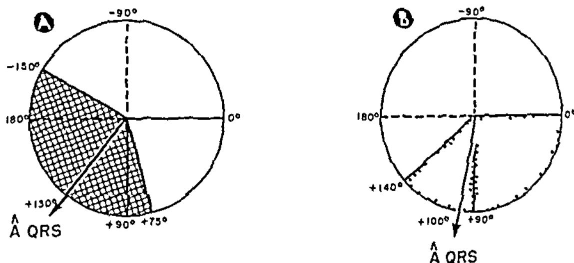


Fig 122 —The extreme range of variation and average orientation of Â QRS in the frontal plane in (A) congenital heart disease (with overloading of the right ventricle) and (B) mitral stenosis

pattern is one that is frequently observed in normal electrocardiograms while the second RSR pattern has been considered in the past to represent incomplete right bundle branch block As previously indicated there is good reason to believe that in many cases the incomplete right bundle branch block pattern actually represents right ventricular hypertrophy In our 21 cases with RSR deflections in lead V_1 the R wave was smaller than 7mm in amplitude in all but 1 case which had an R of 7 mm Thus none of the electrocardiograms fulfilled Barker and Valencia's criteria for the diagnosis of right ventricular hypertrophy in the presence of incomplete or complete right bundle branch block (R' >10 mm or >15 mm amplitude respectively) Moreover 9 of the 21 patients with RSR deflections in lead V_1 failed to present vectorcardiographic evidence of right ventricular hypertrophy

3 In the remaining 30% of the cases lead V_1 displayed QRS complexes of various configurations other than RSR′ (qR qRs Rs R etc) but in [illegible]

all of these cases if an R/S ratio greater than 1 in lead V_1 was the only requirement for making the diagnosis of right ventricular hypertrophy Fifteen electrocardiograms in our series were placed in this category but in only 8 of these did the R wave in V_1 equal or exceed 7 mm In this group of electrocardiograms the onset of the intrinsicoid deflection in lead V_1 occurred later than 0 03 second in almost 65% of the cases

4 Right axis deviation of the mean manifest electrical axis of QRS was present in 45% of the electrocardiograms in the combined series left axis deviation was noted in 5% and no axis deviation in the remaining 50% (Fig 122) Right axis deviation of Â QRS when present was not usually marked

VECTORCARDIOGRAPHIC FINDINGS

The QRS sÊ loop findings in patients with relatively pure mitral stenosis and with mixed mitral stenosis and insufficiency (and or aortic valvular disease) presented below were compiled from the series of Donoso and his co workers Scherlis and his associates and the authors of this text When these series were combined the number of cases of relatively pure mitral stenosis totaled 81 while the total number of cases with mitral stenosis and insufficiency or other valvular lesions was 39

1 In almost 75% of the pure mitral stenosis cases and 25% of the cases with other valvular lesions in addition to mitral stenosis the QRS sÊ loop presented evidence of right ventricular hypertrophy In our 36 cases of pure mitral stenosis 23 vectorcardiograms (64% of the records) were diagnostic of right ventricular hypertrophy 16 showing the RSR pattern of right ventricular hypertrophy and 7 the tall R pattern of right ventricular hypertrophy In the 17 cases of mitral stenosis accom

panied by mitral insufficiency or aortic valve disease 5 vectorcardiograms (29% of the records) were diagnostic of right ventricular hypertrophy and all 5 showed the RSR pattern of right ventricular hypertrophy

2 Left ventricular hypertrophy patterns of the QRS sÊ loop were not observed in any cases of pure mitral stenosis but were present in 22% of the vectorcardiograms of patients with multivalvular lesions

3 Eight of the 43 cases of pure mitral stenosis reported by Donoso and his associates presented the following vectorcardiographic pattern which these investigators believe represents right ventricular hypertrophy The major portion of the QRS loop is located anterior to the isoelectric point although the direction of inscription in the horizontal plane is counterclockwise

4 In their series of mitral stenosis cases Whipple and his co workers observed QRS sÊ loops with posterior and superior terminal appendages without conduction delay in many vectorcardiograms not diagnostic of right ventricular hypertrophy and we have had much the same experience in our mitral stenosis cases (and in cases of chronic cor pulmonale as well) The QRS sÊ loop findings in mitral stenosis without vectorcardiographic right ventricular hypertrophy can be reduced to three loop patterns which for the present will be described only sketchily since these patterns will be discussed in detail in the following chapter on cor pulmonale For descriptive convenience the three QRS sÊ loop patterns will be referred to hereafter as "types A B and C" QRS sÊ loop patterns

Type A QRS sÊ Loop Pattern

The QRS sÊ loop in this pattern (Fig 123) differs from the normal in that it displays a terminal deflection of varying size directed to the right posteriorly and superiorly In the horizontal projection the terminal deflection usually presents a spikelike appearance Unless complicated by some other abnormality neither this QRS sÊ loop pattern nor the other two patterns to be described are characterized by conduction delay

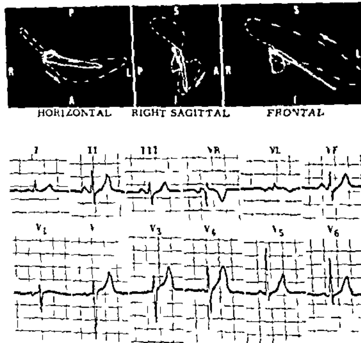


Fig 123 —Type A QRS sÊ loop pattern in mitral stenosis The electrocardiogram is essentially normal The planar QRS loops in the vectorcardiogram are normal in orientation and appearance with the exception of the large terminal deflection of the QRS sÊ loop to the right posteriorly and superiorly

Type B QRS sÊ Loop Pattern

For reasons to be discussed later we believe this QRS sÊ loop pattern (Fig 124) to be very suggestive if not diagnostic of right ventricular hypertrophy As in the type A pattern the QRS [illegible] the type B [illegible] terio[illegible] ceed [illegible] occurring in the former loop pattern Moreover the afferent limb of the QRS

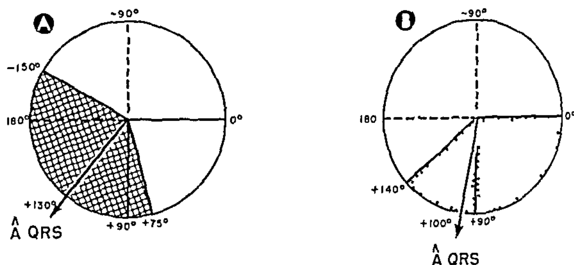


Fig 122 —The extreme range of variation and average orientation of Â QRS in the frontal plane in (A) congenital heart disease (with overloading of the right ventricle) and (B) mitral stenosis

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4 Right axis deviation of the mean manifest electrical axis of QRS was present in 45% of the electrocardiograms in the combined series left axis deviation was noted in 5% and no axis deviation in the remaining 50% (Fig 122) Right axis deviation of Â QRS when present was not usually marked

VECTORCARDIOGRAPHIC FINDINGS

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sÊ loop in the horizontal and sagittal projections occupies a less posterior or more anterior position than [illegible] the type A pattern so that characteristically [illegible] slightly anterior or posterior and [illegible] of the loop is clockwise inscribed far to the right and posteriorly At times the sagittal QRS loop also displays a figure-of eight configuration in which the distal loop of the "eight" is counterclockwise inscribed

Type C QRS sÊ Loop Pattern

This type of QRS sÊ loop pattern (Fig 125) is far less frequently encountered in mitral stenosis than in chronic cor pulmonale and pulmonary emphysema and consists of marked posterior rotation of the entire QRS loop without any striking changes in its appearance otherwise Usually the terminal QRS vectors are directed slightly to the left or right far posteriorly and inferiorly We believe that this QRS sÊ loop pattern reflects more than either of the patterns already described, the effects of clockwise rotation of the heart on its longitudinal axis

The frequency of these various QRS sÊ loop patterns in mitral stenosis the manner in which they are produced and their significance will be dealt with in parallel with the corresponding facts relating to the vectorcardiographic findings in chronic cor pulmonale in the following chapter

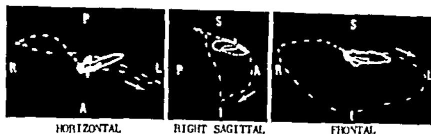
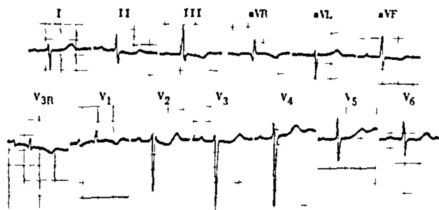


Fig 124 — Type B QRS sÊ loop pat

deflections in leads V3R and V1 are compatible with right ventricular hypertrophy Since the horizontal QRS loop of the vectorcardiogram extends about equally to the left and right ...





trast with the horizontal QRS loop in the type A pattern the type B horizontal QRS loop is characterized by anterior displacement of the left to right limb of the loop which causes the horizontal loop to have a figure-of-eight configuration the first half of the loop being written in a counter clockwise direction to the left and slightly anteriorly and the second half in a clockwise direction to the right and posteriorly Unlike the frontal QRS loop in the type A pattern which is frequently counterclockwise inscribed the frontal loop in the type B pattern as exemplified by the above vectorcardiogram is generally written in a clockwise direction On rare occasions the type B QRS sÊ loop pattern is observed in vectorcardiograms recorded from normal individuals but as a rule when it occurs in patients with mitral stenosis or chronic pulmonary disease it is strongly suggestive of right ventricular hypertrophy

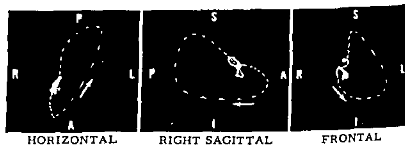
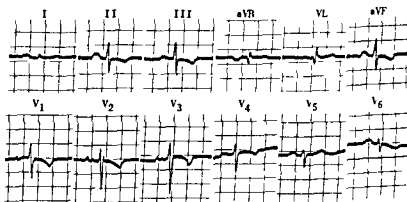


Fig 125 — Type C QRS sÊ loop pattern in mitral stenosis The terminal portion of the QRS sÊ loop in this pattern is located superiorly but the most characteristic feature of the QRS loop is its posterior rotation Significant rightward extension of the terminal part of the QRS sÊ loop in the type C pattern is uncommon in mitral stenosis but a relatively frequent finding in the corresponding QRS sÊ loop pattern in chronic pulmonary disease



or rarely primary pulmonary hypertension. The discussion to follow—and for that matter the greater portion of this chapter—will be devoted to a consideration of the pathophysiology and electrocardiographic and vectorcardiographic manifestations of chronic cor pulmonale, particularly cor pulmonale occurring in patients with chronic obstructive emphysema.

No attempt will be made in this text to discuss the various types of lung disease or pulmonary vascular disease which may lead to cor pulmonale, nor will it be possible to describe all of the pathophysiologic mechanisms concerned. Instead, the discussion will center almost entirely on chronic cor pulmonale due to pulmonary emphysema, although for all intents and purposes the points developed during the discussion have equal relevance to chronic cor pulmonale resulting from other types of chronic lung or pulmonary vascular disease.

The attempt to establish or refute the diagnosis of chronic cor pulmonale is more than just an intellectual exercise serving no practical purpose. Chronic cor pulmonale complicating pulmonary emphysema or some other type of chronic lung disease has important therapeutic and prognostic implications ... of a pa... es proves ... ironic cor pulmonale for several reasons. (1) Neither radiologic evidence of severe chronic emphysema or fibrosis nor catheterization proved pulmonary hypertension can be accepted as proof of the existence of chronic cor pulmonale. (2) The occurrence of congestive heart failure in a patient with chronic lung or pulmonary vascular disease may be related etiologically to some other type of heart disease, such as arteriosclerotic heart disease. (3) Right ventricular enlargement is particularly difficult to detect radiologically in patients with pulmonary emphysema. (However, there is sometimes indirect evidence of cor pulmonale, such as dilatation of the proximal portion of the main pulmonary artery.) Since other clinical studies may give at times equivocal results with reference to the diagnosis of chronic cor pulmonale, it can readily be appreciated that in some instances the electrocardiographic findings are of crucial importance.

GENESIS OF ECG FINDINGS IN CHRONIC COR PULMONALE DUE TO PULMONARY EMPHYSEMA

Chronic pulmonary emphysema may affect the electrocardiogram directly and indirectly in the following ways:

1. Emphysema decreases the conductivity of the lung and thereby impedes transmission of cardiac potentials to the body surface. As previously indicated, the emphysematous lung contains far more air than the normal lung, since there is poor respiratory exchange and trapping of air in the large air sacs formed by overdistended or coalescent alveoli. Because the lung tissue interposed between the heart and body surface contains more air than normally and because air is an extremely poor conductor, the electrical forces arising in the heart are poorly transmitted to the surface electrodes of the electrocardiograph. In this event, the electrocardiogram displays low voltage deflections in all leads. The presence of low voltage is determined chiefly from the size of the QRS deflections, since corresponding criteria for low voltage of the P and T waves are lacking. (The significance of the latter finding would be almost impossible to assess anyway.) Certainly, low voltage QRS deflections are not invariably observed in pulmonary emphysema, and their presence does not necessarily correlate with the severity of the lung disease. In our experience, the more common finding in pulmonary emphysema is low QRS voltage which is limited to the extremity leads. In most cases, this finding can be attributed to the fact that the QRS forces are oriented almost directly posteriorly and therefore lie nearly perpendicular to the frontal plane.

2. Emphysema is accompanied by lowering of the diaphragm, and this in turn causes the heart to descend in the chest, to assume a more vertical position, and to rotate in a clockwise direction around its longitudinal axis. The clockwise rotation causes the left ventricle to be displaced posteriorly and the right ventricle to rotate to a more anterior position. The ... vertical heart position, right axis deviation of $\hat{A}$QRS, and the precordial lead QRS pattern of marked clockwise rotation.

3. Th... can lead ... overloa... ... hypertrophy and dilatation and, in some cases, to cardiac failure. Thus Mounsey and other investigators have observed right ventricular hypertrophy in approximately 60% of the

Cor Pulmonale and Pulmonary Emphysema

THE SYNONYMOUS TERMS *pulmonary heart disease* and *cor pulmonale* are utilized specifically to designate hypertrophy, dilatation and/or failure of the right heart caused by an increased pulmonary vascular resistance, whether the latter is due to intrinsic disease of the lung or to pulmonary vascular disease. The direct cause of cor pulmonale occurring in cases of lung disease or pulmonary vascular disease is pulmonary hypertension; however, not every patient with pulmonary hypertension has cor pulmonale, and so the terms *cor pulmonale* and *pulmonary hypertension* are not equivalent in meaning.

CLINICAL FORMS OF COR PULMONALE

Cor pulmonale may present clinically in three forms—*acute*, *subacute* and *chronic cor pulmonale*—which differ according to the rapidity of onset and duration of the pulmonary hypertension and cor pulmonale.

Acute cor pulmonale.—This form consists of acute and usually transient pulmonary hypertension resulting from sudden embolic occlusion of a major branch of the pulmonary artery or from widespread seeding and occlusion of smaller vessels by many small emboli. Pulmonary arteriospasm may also be an important factor in the genesis of the pulmonary hypertension and right ventricular dilatation. In occasional cases of recurrent pulmonary embolism, chronic cor pulmonale is the eventual outcome and has been attributed to pulmonary arteriosclerosis, which many authors believe is related in some way to organization of the emboli. The electrocardiographic and vectorcardiographic findings in acute cor pulmonale are described later in this chapter.

Subacute cor pulmonale.—Since the subacute type of cor pulmonale is usually encountered in cases of diffuse carcinomatosis of the lungs, it is not surprising that the rapidity of onset and duration of the cor pulmonale are intermediate between the two extremes of acute and chronic cor pulmonale. In about 75% of the cases of diffuse carcinomatosis of the lung reported in the medical literature, the primary tumor was gastric carcinoma. Tumors can spread to the lungs via the blood vessels and/or lymphatics. Retrograde spread of tumor from the hilar lymph nodes to the perivascular lymphatics of the lungs is referred to as *lymphangitic carcinomatosis*.

In 11 of the 78 reported cases of diffuse carcinomatosis of the lung, right ventricular hypertrophy was found at postmortem examination, and in 10 of the 11 cases, according to Morgan, obliterative lesions of the pulmonary arterioles in the form of intravascular fibrosis or more recent thrombosis were observed. Tumor cells were usually demonstrable in the blood vessels as well as in the perivascular lymphatics, and the resulting obliterative endarteritis was held responsible for the pulmonary hypertension and hypertrophy and/or eventual failure of the right ventricle occurring in occasional patients with diffuse carcinomatosis of the lung. Since the electrocardiographic and presumably vectorcardiographic manifestations of this type of cor pulmonale are essentially similar to those occurring in chronic cor pulmonale, and because of the rarity of subacute cor pulmonale, the latter will not be considered individually in this text.

Chronic cor pulmonale.—This form is seen most commonly in cases of long standing obstructive emphysema but may also occur in patients with severe kyphoscoliosis, fibrotic or granulomatous disease of the lungs, chronic multiple pulmonary emboli, m

THE ECG AND VCG FINDINGS IN CHRONIC COR PULMONALE AND IN PULMONARY EMPHYSEMA WITHOUT COR PULMONALE

The principal effects on the heart of the increased pulmonary vascular resistance in chronic cor pulmonale consist of (*a*) right atrial enlargement and (*b*) systolic overloading of the right ventricle which is responsible in whole or in part for such *electrocardiographic abnormalities* as right axis deviation of Â QRS the precordial QRS pattern of "marked *clockwise rotation*" and the fully developed right ventricular enlargement patterns

Right Atrial Enlargement

It will be recalled that the enlargement of the left atrium occurring in mitral stenosis is associated with disturbances of the atrial rhythm the most common *of which* is atrial fibrillation or with the electrocardi

TABLE 13 – CARDIAC RHYTHM IN CHRONIC COR PULMONALE

Study Group	No of Cases		
	Total	Normal Sinus Rhythm	Atrial Fibrillation
Parkinson and Hoyle	80	79	1
Scott and associates	28	27	1†
Authors series	37	36	1‡
Total	145	142	3

A p tient with systemic hyperte

rad
alsc

ographic P wave *pattern* of P mitrale In contrast right atrial enlargement in chronic cor pulmonale rarely produces arrhythmias *although* it frequently is accompanied by a character istic P wave pattern the P pulmonale *pattern* *In fact* the relative rarity of cardiac arrhythmias *in patients* with of ... pulm ...
freq
stand ... use of arteriosclerotic heart disease is considered *In* a combined series of chronic cor pulmonale cases of Parkinson and Hoyle Scott and *his* associates and the authors of this text (Table 13) 142 *of the* 145 cases exhibited *sinus* rhythm the 3 *exceptional* cases showing atrial fibrilla tion Interestingly enough each of the 3 cases with atrial fibrillation had generalized cardiac enlargement on roentgenographic study (*1 patient from each* group of the series making up the combined group) and 1 case was found to have systemic hypertension Thus the demonstration of atrial fibrillation *or some* other cardiac arrhythmia in a case with the clinical diagnosis of chronic cor pulmonale either should raise some question as to the correctness of this diagnosis or should suggest the possibility of *coexisting heart disease* of some other type

The ECG and VCG P Pulmonale Pattern

ELECTROCARDIOGRAPHIC P PULMONALE PATTERN

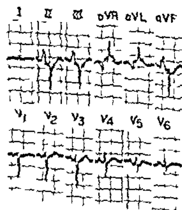
The electrocardiographic features of the P *pulmonale* pattern occurring *in chronic* cor pulmonale (Fig 126) are as follows

1 The mean manifest electrical axis of P (Â P) or the frontal plane *mean* P vector is situated to the right of its average normal location at +60 *in*

Fig 126 – Electrocardiographic findings in chronic cor pulmonale The P pulmonale pattern of right atrial en largement is *evidenced by the following* Â P in the frontal plane located at about +60 and a P wave ampli

does not militate against the diagnosis *of* right atrial en

monale



cases of pulmonary emphysema which they studied. The different mechanisms in pulmonary emphysema which may sometimes increase pulmonary vascular resistance and thereby cause systolic overloading of the right ventricle are as follows:

a) The anatomic restriction of the pulmonary vascular bed may be one factor. Microscopic examination of the emphysematous lung reveals that the alveolar septa in many regions of the lung are atrophic, ruptured, or entirely absent; that many of the alveoli have coalesced to form large air sacs; and finally, that there is rather extensive interstitial fibrosis. These pathologic changes in turn cause compression, narrowing, thrombosis, and obliteration of many of the pulmonary capillaries, with consequent restriction of the pulmonary vascular bed. As a result, the ability of the pulmonary vascular bed to adjust to increases in blood flow becomes greatly limited, and exercise may lead to a rise in pulmonary artery pressure. Eventually, the restriction of the pulmonary vascular bed may become so marked that the pulmonary artery pressure is elevated even at rest.

b) Arterial hypoxemia may be a factor. The characteristic abnormality of respiratory function in pulmonary emphysema is poor aeration of perfused alveoli, which may lead to pronounced arterial hypoxemia and carbon dioxide retention. Systemic arterial hypoxemia in turn may produce in the following: (1) polycythemia and hypervolemia, (2) increased cardiac output and pulmonary blood flow, and (3) possibly pulmonary vasoconstriction. All of these effects of hypoxemia tend to augment or exaggerate the effect of anatomic restriction of the pulmonary vascular bed and therefore tend to produce an even greater degree of pulmonary hypertension.

c) Additional factors, such as bronchopulmonary arterial shunts, pulmonary arteriosclerosis, increased bronchomotor tone, and increased alveolar pressure, probably play less important parts in the genesis of pulmonary hypertension than the factors described above.

From the foregoing, it is evident that the electrocardiographic features of chronic cor pulmonale are a composite of changes due to the mechanical effects of emphysema per se and to superimposed right ventricular hypertrophy, but the dividing line between the two types of electrocardiographic manifestations is exceedingly tenuous. In any event, the important thing is that the electrocardiographic features of right ventricular hypertrophy in chronic cor pulmonale are almost invariably modified by the coexisting abnormalities resulting solely from changes in cardiac position or rotation. More will be said later of the manner in which changes in heart position and cardiac rotation in chronic cor pulmonale influence the orientation and magnitude of the mean instantaneous QRS spatial vectors.

CHRONIC COR PULMONALE AND CHRONIC PULMONARY DISEASE WITHOUT DETECTABLE COR PULMONALE

The electrocardiographic findings in 37 patients hospitalized at Barnes Hospital with chronic lung or pulmonary vascular disease will be described in the following pages (see also Table 15). One or more electrocardiograms and vectorcardiograms were recorded from each patient. For purposes of comparison, the 37 cases were divided into two groups, one group having radiologically demonstrated or autopsy proved cor pulmonale and the other chronic lung disease without detectable cor pulmonale. The two groups will be referred to hereafter as the chronic cor pulmonale group and chronic pulmonary emphysema group, respectively. Even though the second group of cases presented no roentgenographic evidence of cor pulmonale, many of the patients had severe pulmonary emphysema and a third of them were in congestive heart failure at the time of hospitalization. The types of lung disease in the two groups and the number of cases in each group follow:

	No. of Cases
I Chronic cor pulmonale group	20
Pulmonary emphysema with or without fibrosis	15
Fibrocalcific tuberculosis	1
Cystic disease of the lungs	1
Interstitial fibrosis of both lungs	1
Severe kyphoscoliosis	1
Minimal emphysema, pulmonary arteriosclerosis suspected	1
II Chronic pulmonary emphysema group	17
Pulmonary emphysema with or without fibrosis	14
Pectus excavatum	1
Bronchiectasis	1
Pneumoconiosis	1

Patient was a 20 year-old man

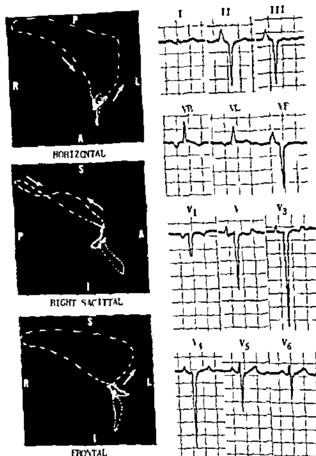


Fig 127 —Electrocardiographic and vectorcardiographic P pulmonale pattern of right atrial enlargement and type A QRS sÊ loop pattern in a man 53 with chronic cor pulmonale (secondary polycythemia arterial oxygen saturation of 76% congestive heart failure and radiologic findings of right ventricular hypertrophy and of pulmonary emphysema and fibrosis)

the frontal reference frame and approaches but with rare exceptions never exceeds +90°

2 The magnitude of the mean instantaneous P vectors and Â P is increased and their average direction as represented by the frontal and horizontal plane mean P vectors tends to be more inferior anterior and less to the left than normally The characteristic orientation of the mean instantaneous P vectors in P pulmonale is thought to reflect the electrical predominance of the enlarged right atrium which is situated anteriorly inferiorly and to the right of the left atrium Since the right atrium is normally the first to undergo activation prolongation of its activation time as the result of dilatation and hypertrophy does not usually cause widening of the P waves but because of the greater magnitude of the P wave forces produced by the right atrium the P waves are of increased amplitude

Our diagnosis of P pulmonale is based on the presence of tall peaked or occasionally slurred or notched P waves of 2.5 mm or more amplitude in leads II III and/or aVF The following findings support the diagnosis of P pulmonale but are not diagnostic in themselves

a) *Diphasic or inverted P waves of increased size* in lead V_1 (a less common finding but one more strongly favoring the diagnosis of P pulmonale *is the presence of tall upright P waves* in lead V_1)

b) Low almost flat P waves in lead I

Occasionally dilatation of a hypertrophied right atrium is said to prevent the appearance of the P pulmonale pattern and the explanation usually given is that the increased mass of intra atrial blood has an abnormally marked short circuiting effect on atrial potentials As a matter of fact the exact mechanism responsible for the P pulmonale pattern remains in doubt It seems unlikely that such a nontransient condition as right atrial hypertrophy could be responsible for this abnormality since the electrocardiographic P pulmonale pattern has been observed to appear relatively fleetingly during acute episodes of pulmonary embolism *status asthmaticus* and acute pulmonary edema The relative importance of right atrial dilatation and changes in anatomic heart position and rotation *in the genesis of the P pulmonale* pattern has not been established as yet Myers has observed this P wave pattern occasionally in normal persons with *asthenic body habitus* and attributes its presence to a vertical heart position and a low diaphragm

TABLE 14 — ORIENTATION OF THE MAXIMAL MEAN INSTANTANEOUS VECTOR OF THE PsÊ LOOP IN CHRONIC COR PULMONALE AND CHRONIC PULMONARY EMPHYSEMA WITHOUT COR PULMONALE

	Total No. of Cases	% with VCG P Pulmonale	Horizontal			Right Sagittal			Frontal		
			Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
Chronic cor pulmonale	20	82	−20 to +90	+60	+10 to +80	+50 to +90	+70		+50 to +90	+80	
Chronic pulmonary emphysema without cor pulmonale	17	47	−10 to +80	+35	0 to +60	+60 to +95	+75		+50 to +90	+75*	
(Normal cases)	(60)	(0)	(−20 to +30)	(+5)	(0 to +5)	(+60 to +100)	(+80)	(+80 to +90)	(+10 to +90)	(+55)	(+20 to +60)

The normal values for PsÊ loop orientation are enclosed in parentheses and are the same as those presented in Table 4 in Chapter 7. They are included in the table to provide a basis for comparison with the values obtained in chronic cor pulmonale and chronic pulmonary emphysema without cor pulmonale.

grees of right axis deviation of Â QRS (Fig 128 right) In only 1 of our cases of chronic cor pulmonale was Â QRS situated to the left of +90 In the electrocardiogram in this instance Â QRS was oriented along the −85 axis of the frontal reference frame lead I recorded an Rs deflection of low voltage and leads II and III displayed relatively deep QS deflection (there was no clinical history suggestive of myocardial infarction) However the same electrocardiogram showed the precordial lead QRS pattern of marked clockwise rotation" lead V_6 registering an rS deflection This would indicate that the horizontal plane mean QRS vector was situated posteriorly and to the right of −90 The probable explanation for this discrepancy between the QRS configuration in leads I and V_6 is that in this case the lead vector or effective axis of lead I is canted downward in a left to-right direction while the effective axis of lead V_6 is slanted in just the opposite direction Thus as Schaffer has pointed out a cardiac vector directed almost vertically superiorly will be recorded in lead I as if rotated to the left and in V_6 as if rotated to the right of its actual position In the above exceptional case mean instantaneous QRS spatial vectors appearing during the second half of the QRS interval were probably directed superiorly and somewhat to the right but because of the rotation of the effective axis of lead I they projected on the positive half of the axis of derivation of this lead and therefore produced terminal QRS positivity rather than negativity In support of this explanation is the fact that the frontal QRS loop of the patient's vectorcardiogram showed a maximal mean instantaneous QRS vector oriented along the −120 axis of the frontal reference frame Consequently in this case as probably also in similar cases of left axis deviation in chronic cor pulmonale reported in the past the apparent left axis deviation is actually marked right axis deviation

The S_I-S_{II}-S_{III} pattern consists of predominantly downwardly directed QRS deflections (rS qrS QS) in leads I II and III and is associated with the precordial lead QRS pattern of marked clockwise rotation in which the transitional lead registering the transitional equiphasic RS deflection lies to the left of lead V_4 There is a relatively high degree of correlation between the S_I-S_{II}-S_{III} pattern in the electrocardiogram and the presence of anatomic right ventricular hypertrophy It is this pattern especially which is so frequently interpreted as indicating anterior anteroseptal diaphragmatic or combined infarctions In fact the usual QRS criteria for the diagnosis of myocardial infarction are of limited value in evaluating electrocardiograms presenting other evidence of chronic cor pulmonale

Precordial QRS Pattern of "Marked Clockwise Rotation"

In the absence of electrocardiographic evidence of right ventricular hypertrophy the precordial QRS pattern most characteristic of chronic cor pulmonale is that of "marked clockwise rotation" As will be recalled in the normal electrocardiogram the precordial lead which records the transitional equiphasic RS deflection is usually V_4 or some lead to the right of this point thus the horizontal plane mean QRS vector is normally situated at or anterior to −30 in the horizontal reference frame In chronic cor pulmonale the mean QRS vector is often rotated as far posteriorly as −90° or more and the equiphasic RS deflection is recorded by lead V_5 or some lead to the left of this point As will be described later this electrocardiographic pattern is frequently associated with [illegible]

tion of the heart about its longitudinal axis but a somewhat different mechanism may also be involved in cases with the electrocardiographic pattern of [illegible]

[illegible] cor pulmonale

Electrocardiographic Right Ventricular Hypertrophy Patterns

As will be noted in Table 15 showing the incidence of the various electrocardiographic findings in chronic cor pulmonale the diagnosis of right ventricular hypertrophy was made in only 16–28% of the cases of chronic cor pulmonale in the several series despite the fact that in our series at least the criteria used in making the diagnosis were far from strict Some of the reasons for the failure of the electrocardiogram to detect right ventricular hypertrophy more frequently than it does in chronic cor pulmonale have already been considered and will not be repeated

When right ventricular hypertrophy was diagnosable electrocardiographically in our [illegible] of two QRS [illegible] —namely [illegible] or a QR [illegible] es of chronic cor pulmonale cases 4 out of 20 electrocardiograms showed right ventricular hypertrophy patterns with two showing each of the above types of QBS configura

According to Wood the tall P wave of the P pulmonale pattern is probably the earliest sign of cardiovascular disturbance resulting from emphysema or at least competes in this respect with elevation of the right ventricular pressure and slight reduction of arterial oxygen saturation it may develop several *years before the onset of heart failure* Wood could find no correlation between the presence of the P pulmonale pattern in chronic cor pulmonale and such factors as anoxia cardiac output or right atrial pressure although he felt that there was some relationship between this electrocardiographic finding and right ventricular pressure

It is of some interest that Zuckermann and his coworkers concluded from their studies of patients with chronic cor pulmonale that a small or absent P wave in lead aVF excludes the possibility of chronic cor pulmonale more readily than a tall P wave in this lead establishes the same diagnosis Moreover they state that *when the voltage of the P waves in lead II* or lead III is below 0 5 mm the possibility of uncomplicated chronic cor pulmonale is practically excluded

Vectorcardiographic P Pulmonale Pattern

Comparatively few vectorcardiographic studies of chronic cor pulmonale have been reported in the medical literature The following description of the vectorcardiographic findings in this condition is based in large part on our observations in 37 cases of chronic lung or pulmonary vascular disease with and without chronic cor pulmonale In 20 of the 37 cases there was postmortem proof or roentgenographic evidence of cor pulmonale

The frequency of the vectorcardiographic finding of P pulmonale pattern in our study of cases of chronic cor pulmonale and of cases of chronic lung disease without roentgenographic cor pulmonale and the average orientation and range of variation in orientation of the P sÊ loop are shown in Table 14

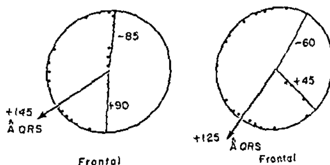
The P sÊ loop abnormalities corresponding to the electrocardiographic pattern of P pulmonale (Fig 127) are as follows

1 The maximal instantaneous P vector or long axis of the P sÊ loop is of increased magnitude
2 In the horizontal *projection the P sÊ loop* frequently shows a figure of eight configuration which differs from that in mitral stenosis in that the initial anterior loop of the eight is larger than the later posterior component The two portions of P sÊ loops with a figure of eight configuration can be inscribed in a counterclockwise-clockwise direction respectively or just the reverse Occasionally a horizontal P loop with a trapezoid like configuration is written entirely in a clockwise direction The corresponding sagittal and frontal P loops can be written in either a clockwise or a counterclockwise direction However the more *common finding is that a P loop with a trapezoid* like configuration in the horizontal projection is entirely counterclockwise inscribed
3 The sagittal P loop in P pulmonale is generally elongated but frequently presents a bifid configuration with the initial and larger portion of the loop lying anteriorly and the remainder of the loop situated posteriorly Occasionally the sagittal P loop has a figure of eight configuration The direction of inscription of the sagittal P loop is usually normal—*that is clockwise*
4 The frontal P loop tends to resemble the sagittal loop in configuration and to be directed almost vertically downward The loop is usually written in a counterclockwise direction
5 As Sano and his associates have pointed out the P sÊ loop in P pulmonale frequently remains open in all three projections representing a displacement of the junction of P with the Ta component

Right Axis Deviation of Â QRS

Chronic cor pulmonale is one of the commonest causes of moderate to marked degrees *of right* axis deviation of Â QRS unaccompanied by more obvious evidence of right ventricular hypertrophy (Fig 128 *left*) while *chronic pulmonary* emphysema without cor pulmonale tends to be accompanied by lesser de-

Fig 128 —Extreme range of variation and average orientation of Â QRS in the frontal plane in chronic cor pulmonale **(left)** and chronic pulmonary emphysema (with*out evident cor pulmonale)* **(right)**



The outflow tract of the right ventricle and the crista supraventricularis is normally one of the last regions of the ventricles to undergo activation and this fact has

the R usually is about the same size as the initial R wave and is said not to exceed 4 mm Actually many of the qR deflections recorded in lead V_1 in patients with chronic cor pulmonale or mitral stenosis are probably equivalent to rsR deflections in which the initial portion of the QRS complex is isoelectric This certainly does not apply in every case however for the vectorcardiograms obtained in some patients display initial deflections of the QRS sÊ loop to the left and posteriorly The explanations currently proposed for this finding have already been discussed in Chapter 10 dealing with the general diagnostic features of right ventricular hypertrophy ... the electrocardiogram and vectorcardiogram Parenthetically it might be added that in all probability the high percentages of cases of chronic cor pulmonale with incomplete right bundle branch block patterns in lead V_1 reported by Scott and his co-workers and by Kilpatrick include many cases in which this electrocardiographic pattern is actually produced by a QRS sÊ loop right ventricular hypertrophy pattern of the RSR type

Right Ventricular Strain Pattern

Many investigators have placed considerable emphasis on the diagnostic finding of a right ventricular *strain pattern* in the electrocardiogram of patients with chronic cor pulmonale We have found this feature to be of little practical value in the electrocardiographic diagnosis of *either chronic cor pulmonale* or right ventricular hypertrophy and the *same experience has been reported by others* In approximately 60% of the cor pulmonale cases and 35% of the cases with chronic lung disease without cor pulmonale there was either slight S–T segment depression or shallow T wave inversion in leads II III and aVF when these leads recorded primarily upright QRS deflections However the ST–T abnormalities in themselves are nonspecific so far as the diagnosis of cor pulmonale ... concerned

lead V_1 If one limits the diagnosis of right ventricular strain to electrocardiograms with inverted T waves in leads V_1 through V_3 only 20% of the cor pulmonale patients and about 6% of the patients with chronic lung disease without roentgenographic evidence of cor pulmonale showed this finding and in some of these instances the T wave inversion extended as far to the left as V_5 or V_6 which would indicate more generalized anterior myocardial ischemia In short we rarely use ST–T wave abnormalities to establish ... what ... The ... of such measurements as the time of onset of intrinsicoid deflection *in* lead V_1 the amplitude of the R wave *in* lead aVR amplitude criteria pertaining to various combinations of R waves in right precordial leads and S waves in left precordial leads etc

VECTORCARDIOGRAPHIC QRS sÊ LOOP PATTERNS IN COR PULMONALE AND PULMONARY EMPHYSEMA

Type A QRS sÊ Loop Pattern

The type A QRS sÊ loop pattern was noted in approximately one quarter of the chronic cor pulmonale cases and in over half of the emphysema cases without radiologic evidence of cor pulmonale

Ho...
is ...
as ...
flec... and of brief duration In rare instances the QRS loop is written almost straight anteriorly and slightly to the left or to the left and posteriorly The efferent limb of the horizontal QRS loop is next inscribed to the left and anteriorly and at about the time the loop reaches its maximal leftward extent it turns in a counterclockwise direction posteriorly The afferent limb of the loop ...

... instead the terminal part of the loop is written as a spikelike deflection far posteriorly and either slightly or far to the right The QRS sÊ loop in this and the other ...

TABLE 15 — ELECTROCARDIOGRAPHIC FINDINGS IN CHRONIC COR PULMONALE*

	SCOTT AND ASSOCIATES	WOOD	KILPATRICK	ZUCKERMAN AND ASSOCIATES	AUTHORS' SERIES	
					Chronic Cor Pulmonale	Chronic Pulmonary Emphysema
No of cases	28	100	20	50	20	17
P pulmonale pattern in bipolar limb leads	39%	85%	60%	28%	50%	18%
Av orientation of Â P				+71°	+72°	+69°
Right axis deviation of Â QRS		46%	85%	96%	80%	20%
Av orientation of Â QRS					+145	+125
S_I–S_{II}–S_{III} pattern		9%	20%	22%	20%	12%
Low QRS voltage†		40%	15%		40%—in limb leads only	12%—in limb lead only and 6%—in limb and precordial leads
Precordial QRS pattern of marked clockwise rotation		16%	40%	60%	75%	59%
ECG right ventricular hypertrophy	28 5%	16%	25%		20%‡	18%
Right bundle branch block						
Incomplete	28 5%	0	20%		0	0
Complete	3 6%	4%	0		0	0

* Data obtained from study series of cases of chronic cor pulmonale reported by the investigators cited in the column headings

† In the reports of Wood and of Kilpatrick no information is given as to the specific leads of the electrocardiogram showing low QRS voltage

‡ The lead V_1 criteria used by the authors of this text in making the electrocardiographic diagnosis of right ventricular hypertrophy in adult patients with chronic cor pulmonale mitral stenosis and certain types of congenital heart disease such as interatrial septal defects are as follow a QRS interval f 0 1 [illegible] or exceeding 1 irrespective of the absc [illegible] which the R is obviously taller than [illegible] R is at least half the size of the S [illegible] (3) an RS deflection in lead V_1 in which the R wave approaches equals or exceeds the S wave in size and is of 0 04 second or longer duration

tion Of the 17 patients with chronic pulmonary emphysema without radiographic evidence of cor pulmonale 2 had rsR deflections in lead V_1 of their electrocardiograms (These 2 cases in all likelihood belong in the chronic cor pulmonale group) In all 6 cases with QR or RSR deflections in lead V_1 the R/S or R /S ratio in this lead equaled or exceeded 1 The average amplitude of the R waves of the QR deflections and the R waves of the RSR complexes was 4 6 mm

As in many electrocardiograms recorded from patients with mitral stenosis the electrocardiographic evidence of right ventricular hypertrophy in chronic cor pulmonale even when present is far from conspicuous probably for the same reasons that the electrocardiogram is less frequently diagnostic of right ventricular hypertrophy in these two types of heart disease than in congenital heart disease The late terminal upright deflection in V_1 which is usually the only evidence of right ventricular hypertrophy in chronic cor pulmonale and is the commonest right ventricular hypertrophy pattern found in mitral stenosis is probably attributable to the selective type of right ventricular hypertrophy present Thus in chronic cor pulmonale and in mitral stenosis the earliest evidence of anatomic hypertrophy of the right ventricle consists of thickening of the muscle around the base of the outflow tract particularly the crista supraventricularis and thickening of the trabeculae carneae and papillary muscles of the right ventricle

emphysema (Fig 129) The average orientation of the maximal leftward mean instantaneous QRS vector was 0 in our cases of mitral stenosis −35° in the chronic cor pulmonale cases and −15 in the chronic emphysema cases By the same token the maximal terminal mean instantaneous QRS vector (the largest instantaneous vector situated to the right of the electrical null point) was located less posteriorly in mitral stenosis (average orientation −145) than in chronic cor pulmonale (average orientation −120) or chronic emphysema (average orientation −125) While in an occasional mitral stenosis case with this type of QRS sE loop pattern the maximal terminal mean instantaneous QRS vector may approach in magnitude the maximal leftward mean instantaneous QRS vector this situation is observed far more frequently in the vectorcardiograms of patients with chronic cor pulmonale and/or pulmonary emphysema

Right sagittal QRS loop —In our cases of mitral stenosis with the type A pattern the sagittal QRS loop tended on the whole to be oriented almost vertically inferiorly the maximal mean instantaneous QRS vector lying on the average along the +105 axis of the sagittal reference frame The QRS loop usually displayed terminally a well-defined and prominent superior deflection the maximal superior mean instantaneous QRS vector having an average orientation of −125

The sagittal QRS loop in our cases of chronic cor pulmonale and pulmonary emphysema showed far more variability in appearance and orientation than in our cases of mitral stenosis Thus while sagittal loops of the type just described were sometimes observed in the chronic cor pulmonale—emphysema group the following variant loop patterns were also frequently encountered

1 A figure-of eight sagittal loop the proximal part of which was counterclockwise inscribed and the distal portion clockwise inscribed In sagittal loops of this type the long axis of the loop was usually directed almost straight posteriorly ranging in orientation between +150 and −150

2 A long narrow sagittal loop inscribed entirely in a counterclockwise direction Sagittal loops of this type were less frequently observed by us than were the previous type but when present —

3

... tal reference frame and a maximal superior mean instantaneous vector located at about −170 Generally the terminal part of a sagittal QRS loop of this type although situated superiorly does not form a well-defined deflection but simply is a continuation of the main body of the loop

Frontal QRS loop —In our cases of mitral stenosis with the type A QRS sE loop pattern the frontal QRS loop was usually counterclockwise inscribed only occasionally clockwise inscribed The maximal leftward mean instantaneous QRS vector in these cases had an average orientation of +30 while the average orientation of the maximal terminal mean instantaneous QRS vector was about −135 in the frontal reference frame On the other hand there was an approximately equal distribution of clockwise and counterclockwise-inscribed frontal loops in the vectorcardiograms of the chronic cor pulmonale cases In the chronic cor pulmonale cases the maximal leftward and maximal terminal mean instantaneous QRS vectors were oriented on the average along the −25 and −155 axes of the frontal reference frame respectively while the average orientations of the corresponding vectors in the emphysema group were +10 and −150 respectively

We are not certain as to the significance of this QRS sE loop pattern but are inclined to believe that in its fully developed form with a prominent right posterior and superior terminal deflection it may represent an early stage in the development of a more typical right ventricular hypertrophy QRS sE loop pattern While it is true that the type A QRS sE loop pattern is sometimes observed in the vectorcardiograms of normal subjects it is equally true that the frequency of this pattern varies inversely with the patient s age Thus the very frequency with which the type A pattern is encountered in early childhood would seem to argue in favor of its being a pattern of right ventricular p ...

In ...

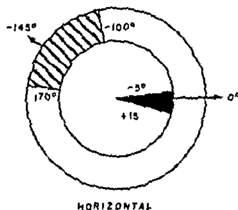
cor p ... this vector cardiographic pattern the electrocardiogram showed varying degrees of right axis deviation in the limb leads and "clockwise rotation" in the precordial leads It is evident that the terminal S waves in the left precordial leads in these cases do not reflect posterior rotation of the QRS sE loop itself but rather are attributable to the rightward terminal deflection of the loop This is to be contrasted with the precordial lead QRS pattern of "clockwise rotation" observed with the type C QRS sE loop pattern (to be described shortly) for in the latter instance the QRS pattern in the chest leads is clearly related to the posterior rotation of the QRS sE loop as a whole Interestingly

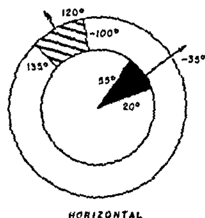
TYPE A QRS sE LOOP PATTERN

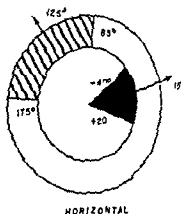
MITRAL STENOSIS (WITHOUT VECTORCARDIOGRAPHIC RVH)

CHRONIC COR PULMONALE (WITHOUT VECTORCARDIOGRAPHIC RVH)

CHRONIC PULMONARY DISEASE (WITHOUT VECTORCARDIOGRAPHIC RVH OR COR PULMONALE)



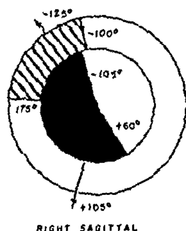


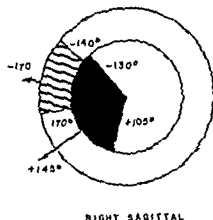


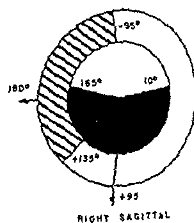
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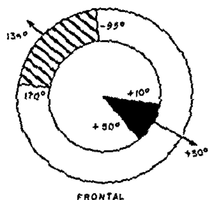


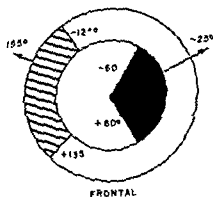


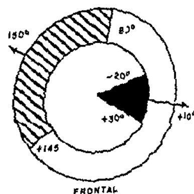
RIGHT SAGITTAL

RIGHT SAGITTAL

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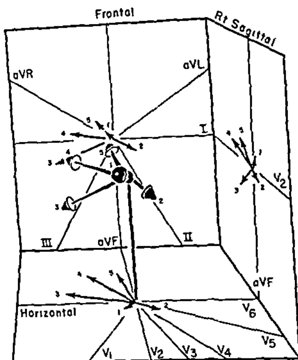
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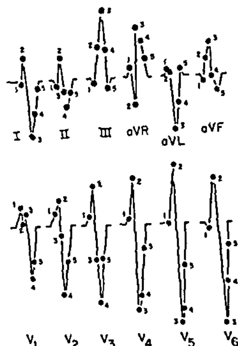
MAXIMAL INITIAL .04 SEC INSTANTANEOUS QRS VECTOR

MAXIMAL TERMINAL .04 SEC INSTANTANEOUS QRS VECTOR

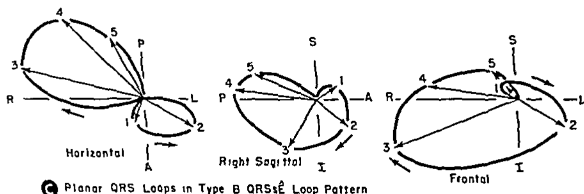
– Average orientation of the maximal initial and terminal 0.04 second mean QRS sE loop pattern in mitral stenosis, chronic cor pulmonale and chronic pulmonale



A Instantaneous VA Vectors in the Type B QRSsÊ Loop Pattern in Mitral Stenosis and Chronic Cor Pulmonale



B QRS Deflections Projected on Scalar Leads



C Planar QRS Loops in Type B QRSsÊ Loop Pattern

Fig 130 —Instantaneous VA vectors in the type B QRS sÊ loop pattern in mitr l t no l nd to ch – nale Note in A that the rightward VA the posterior orientation of these larger r cording their planar projections or is significance of the large rightward late A c tors remains the same—that is they are indicative of right ventricular hypertrophy In B the QRS d flections projected on the scalar leads by the VA vectors exhibit right axis deviation of Â QRS and the precordial QRS pattern of "marked clockwise rotation"

enough in about one half of our cases of mitral stenosis and about one third of our cases of chronic cor pulmonale emphysema lead V_1 of the electrocardiogram recorded an RSR deflection The secondary R wave in these cases presumably corresponds to the terminal rightward deflection of the QRS sÊ loop which evidently projects on the positive half of the axis of derivation of lead V_1 Moreover in approximately one half of all of our chronic cor pulmonale cases with RSR deflections in lead V_1 the R/S amplitude ratio in this lead exceeded 1 The average amplitude of the R wave in these cases was 2 5 mm although the range of variation extended from 0 7 mm to 5 mm

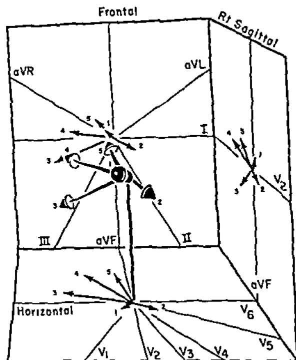
Type B QRS sÊ Loop Pattern

The type B QRS sÊ loop pattern (Fig 130) was observed by us in only 6% of the vectorcardiograms of patients with mitral stenosis (i e only one third as frequently as the type A pattern) while the incidence of this pattern in the chronic emphysema cases was approximately twice as great The type B pattern occurred most frequently in our cases of chronic cor pulmonale and was present in about 30% of the vectorcardiograms recorded In fact this and the type C QRS sÊ loop pattern (see p 209) were the two QRS sÊ loop patterns most commonly encountered in chronic cor pulmonale—at least in our experience

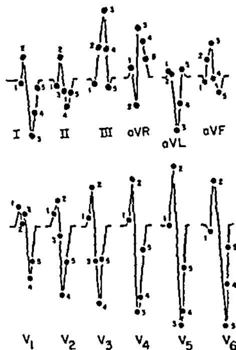
Horizontal QRS loop —In the type B loop pattern the horizontal QRS loop ordinarily exhibits a well developed initial anterior and rightward deflection however this is not invariably true since occasionally the horizontal loop will be found to proceed initially to the left and either anteriorly or posteriorly In either case the efferent limb of the loop is next written to the left and slightly anteriorly or posteriorly although it usually does not extend as far laterally as the corresponding limb of the normal or type A horizontal loop An anterior concavity in the efferent limb of the horizontal QRS loop is not an uncommon finding in vectorcardiograms showing the type B pattern On reaching *its maximal leftward extent* the horizontal QRS loop turns posteriorly and the afferent limb is next inscribed rapidly in a counter clockwise direction from left to right A characteristic feature of the horizontal loop in the type B QRS sÊ loop pattern is that the afferent limb of the loop is written only slightly posterior to the efferent limb and not infrequently curves slightly anteriorly late in its rightward course Soon thereafter the loop turns posteriorly and it continues for a time to be inscribed in a clockwise direction far to the right and far posteriorly before returning eventually to its point of origin In short the horizontal QRS loop in the type B pattern typically presents a figure-of eight configuration in which the early leftward loop of the eight" is counterclockwise inscribed and the terminal rightward and posterior loop is clockwise inscribed Generally the terminal rightward and posterior component of the horizontal loop is larger and encloses a greater area than the earlier leftward component this being especially true in vectorcardiograms of patients with chronic cor pulmonale The average orientation of the maximal leftward mean instantaneous QRS vector in our cases of mitral stenosis was +10° in chronic cor pulmonale −10 and in chronic emphysema +5 and the average orientation of the maximal terminal mean instantaneous QRS vector was respectively −165° −125 and −115 (Fig 131)

Right Sagittal QRS loop —The sagittal loop in the type B pattern sometimes resembles that in the type A pattern but more frequently shows a triangular configuration In other words the sagittal loop displays two distinct maximal mean instantaneous vectors The earlier of the two maximal mean instantaneous QRS vectors is usually directed inferiorly and slightly posteriorly while the terminal maximal vector is oriented slightly superiorly and almost directly posteriorly Depending on the magnitude and orientation of these two vectors the limb of the loop extending between them is written either far posteriorly in which case the sagittal loop is entirely clockwise inscribed or more anteriorly In the latter event the sagittal loop may present a figure of eight configuration with the distal loop of the eight situated slightly anteriorly and having a counterclockwise direction of inscription while the remainder of the loop is written in a clockwise direction slightly posteriorly By and large in our cases of mitral stenosis chronic cor pulmonale and emphysema with the type B QRS sÊ loop pattern the average orientation of the earlier of the two maximal mean instantaneous vectors of the sagittal QRS loop varied between +80 and +115 while the average orientation of the second maximal vector ranged between 180 and −155 In general the early maximal instantaneous QRS vector tended to be directed more posteriorly and the terminal maximal vector more superiorly in the chronic cor pulmonale cases than in the mitral stenosis or chronic emphysema cases

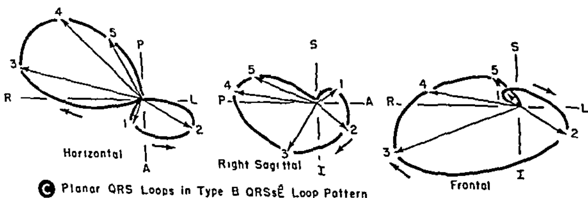
Frontal QRS loop —The frontal loop in the type B QRS sÊ loop pattern usually extends about equally first to the left and then to the right of the electrical null point In over 75% of the vectorcardiograms show



Ⓐ Instantaneous VA Vectors in the Type B QRSsÊ Loop Pattern in Mitral Stenosis and Chronic Cor Pulmonale



Ⓑ QRS Deflections Projected on Scalar Leads



Ⓒ Planar QRS Loops in Type B QRSsÊ Loop Pattern

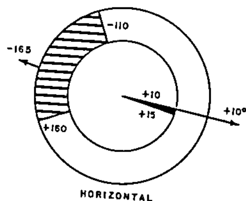
Fı 130 –In t nt n m VA ... nale
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A QRS and the precordial QRS pattern o

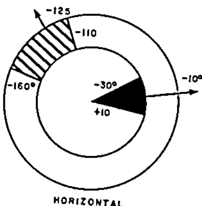
TYPE B QRS sÊ LOOP PATTERN

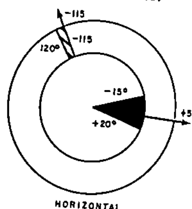
MITRAL STENOSIS (WITHOUT VECTORCARDIOGRAPHIC RVH)

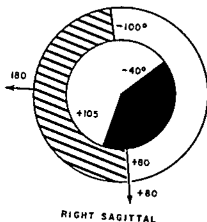
CHRONIC COR PULMONALE (WITHOUT VECTORCARDIOGRAPHIC RVH)

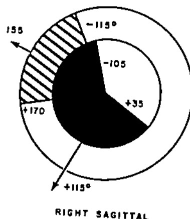
CHRONIC PULMONARY DISEASE (WITHOUT VECTORCARDIOGRAPHIC RVH OR COR PULMONALE)

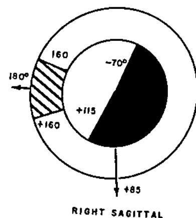


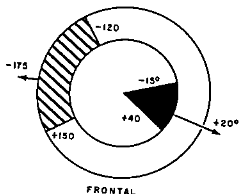


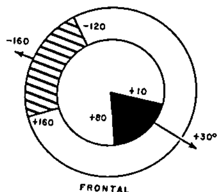


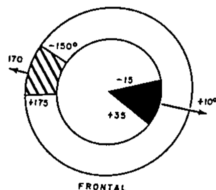












MAXIMAL INITIAL 04 SEC INSTANTANEOUS QRS VECTOR

MAXIMAL TERMINAL 04 SEC INSTANTANEOUS QRS VECTOR

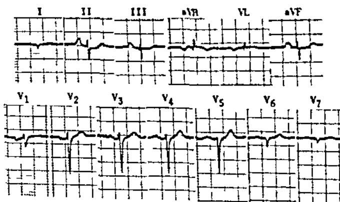
Fig 131 —Extreme range of instantaneous QRS vectors in the monary emphysema without evic

ing this pattern we found the frontal loop to have a clockwise direction of inscription while in less than a quarter of the cases a counterclockwise-inscribed frontal loop was noted Occasionally the frontal loop may have a figure-of-eight configuration with a counterclockwise-inscribed leftward component and a clockwise inscribed rightward component The average orientation of the maximal leftward mean instantaneous QRS vector in our cases of mitral stenosis

ject but such an event has been observed so rarely in our experience as to constitute an exception to the rule The reasons for our believing that the type B pattern reflects right ventricular hypertrophy are

cases of mitral stenosis −1/0 in chronic cor pu





deflections in precordial leads V through V are directed primarily downward Â QRS in the horizontal plane is located to the right and posteriorly A rightward posterior and superior orientation of sÂ QRS as calculated from the electrocardiographic leads correlates very well with the presence of chronic cor pulmonale In the vectorcardiogram the P sÊ loop is larger than normal and is situated abnormally anteriorly and almost vertically inferiorly These findings are consistent with the vectorcardiographic diagnosis of P pulmonale The QRS sÊ loop is written very briefly to the left but soon turns and is written to the right posteriorly and superiorly The configuration of the QRS sÊ loop is compatible with the type B pattern of right ventricular hypertrophy The T sÊ loop is discordant to the long axis of the QRS sÊ loop

chronic cor pulmonale and chronic emphysema ranged between +10 and +30 while the average orientation of the maximal rightward mean instantaneous vector varied between −175 and −160 (Fig 131)

It is our belief that the type B QRS sÊ loop pattern when observed in cases presenting other clinical findings suggestive of mitral stenosis or chronic cor pulmonale

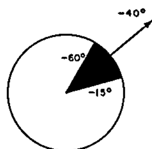
monale and +125 in chronic emphysema By and

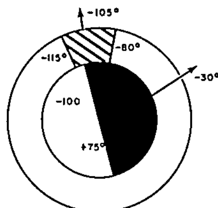
played more marked right axis deviation of Â QRS than in the cases in which the vectorcardiograms showed the type A loop pattern Although in many cases the QRS deflections in the bipolar limb leads approached the S_I–S_{II}–S_{III} configuration in appearance we observed the latter pattern only in the chronic cor pulmonale cases but in this group the pattern appeared in approximately one half of the

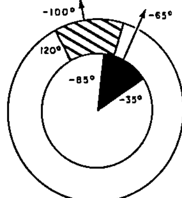
MITRAL STENOSIS (WITHOUT VECTORCARDIOGRAPHIC RVH)

CHRONIC COR PULMONALE (WITHOUT VECTORCARDIOGRAPHIC RVH)

CHRONIC PULMONARY DISEASE (WITHOUT VECTORCARDIOGRAPHIC RVH OR COR PULMONALE)



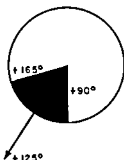


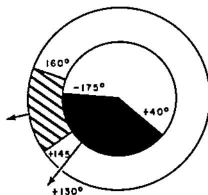


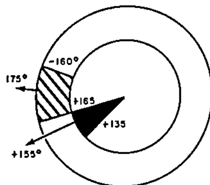
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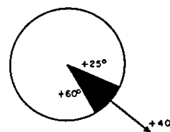


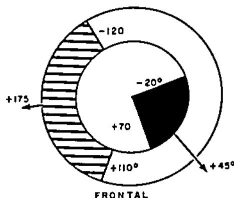


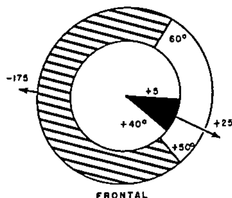
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RIGHT SAGITTAL







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■ MAXIMAL INITIAL .04 SEC INSTANTANEOUS QRS VECTOR

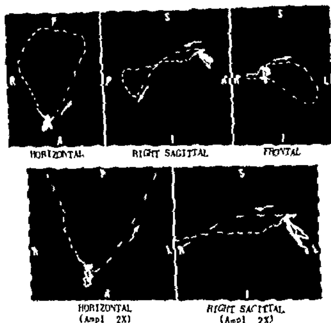
▧ MAXIMAL TERMINAL .04 SEC INSTANTANEOUS QRS VECTOR

Fig 133 —Extreme range of variation and average orientation of the maximal initial and terminal 0 04 second mean instantaneous QRS vectors in the type C QRS sÊ loop pattern in mitral stenosis chronic cor pulmonale and chronic pulmonary emphysema without cor pulmonale

electrocardiograms In addition the electrocardiograms of most of the chronic cor pulmonale patients and of slightly fewer chronic emphysema patients showed the P pulmonale pattern The QRS configuration in lead V_1 in our cases of mitral stenosis chronic cor pulmonale and chronic emphysema usually was of a vibratory rsr's qR rSr' or rS type Moreover the precordial leads almost invariably displayed the

Type C QRS sÊ Loop Pattern

The type C QRS sÊ loop pattern appeared in about the same percentage of vectorcardiograms of our patients with mitral stenosis and chronic cor pulmonale as did the type B pattern although it occurred much more frequently than the latter pattern in the cases of chronic emphysema where it was present in



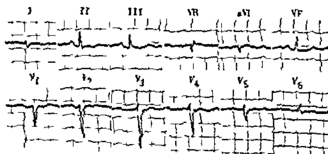


Fig 134 —Vectorcardiographic type C QRS sÊ loop pattern in a man 54 with severe obstructive emphysema and congestive heart failure but *without radiologic evidence of cor pulmonale* The right axis deviation of Â QRS in the frontal plane scalar leads and the marked posterior rotation of Â QRS in *the horizontal scalar leads are compatible* with chronic pulmonary disease The entire QRS sÊ loop of the vectorcardiogram is rotated abnormally far posteriorly so that the long axis of the horizontal QRS loop is *directed along the* −90 axis of the horizontal reference *frame The T* sÊ loop is discordant to *the QRS* sÊ loop and is oriented almost directly anteriorly

electrocardiographic pattern of "marked clockwise rotation" In occasional electrocardiograms Q waves of such prominence as to raise the question of myocardial infarction were recorded in leads II III or aVF or in one or more of the right precordial leads but in none of these cases was there historical or other clinical evidence suggesting antecedent infarction Thus one should be wary of making the erroneous electrocardiographic diagnosis of myocardial infarction in tracings compatible with chronic cor pulmonale

almost one third of the vectorcardiograms studied

Horizontal QRS Loop —There were certain differences in the appearance of the QRS sÊ loop in the type C pattern in chronic cor pulmonale and emphysema as compared with the pattern in mitral stenosis The dissimilarities were usually most striking in the horizontal projection For example in our few cases of mitral stenosis in which the vectorcardiograms displayed the type C QRS sÊ loop pattern the horizontal QRS loop presented an essentially normal configuration but its long axis or maximal mean in

stantaneous vector was simply rotated abnormally far posteriorly, *its average orientation being about −40*. In contrast, the horizontal QRS loop in the chronic cor pulmonale and chronic emphysema cases displayed the following features:

1. The initial deflection of the loop was written almost straight anteriorly or sometimes slightly to the right or left and anteriorly.
2. The loop then turned abruptly posteriorly, and the efferent limb of the loop was written as an almost straight line far posteriorly and to a varying degree to the left.
3. On reaching its maximal leftward and posterior extent, the loop then turned medially and proceeded rapidly from left to right.
4. The afferent limb and terminal part of the loop were often written in the form of a large rightward and posterior deflection, much like *that occurring in* the fully developed type A QRS sÊ loop pattern, resemble that occurring in the type A pattern, although generally being oriented somewhat more inferiorly. If the frontal QRS loop should happen to be oriented almost vertically inferiorly, then the horizontal QRS loop *may simulate that observed in the* type A QRS sÊ loop pattern; however, this is simply the result of foreshortening of the loop in the horizontal projection (Fig. 134 and Table 16).

We are not certain as to the manner of production or the significance of the type C QRS sÊ loop pattern, although we have observed 2 patients with chronic cor pulmonale with this pattern who were found at postmortem to have marked right ventricular hypertrophy. Although there is no other evidence to support this impression, we nonetheless feel that the type C pattern as it appears in the chronic cor pulmonale cases—that is, with large rightward, posterior *and superior terminal mean instantaneous QRS vectors* in addition to the abnormal posterior rotation of

TABLE 16.—QRS sÊ Loop Patterns in Mitral Stenosis, Chronic Cor Pulmonale and Chronic Pulmonary Disease without Cor Pulmonale

	Total No. of Cases	Normal QRS sÊ	Right Ventricular Hypertrophy	Left Ventricular Hypertrophy	Type A QRS sÊ	Type B QRS sÊ	Type C QRS sÊ
Mitral stenosis	54	3	32	4	9	3	3
Chronic cor pulmonale	20	0	3	0	5	6	6
Chronic pulmonary disease	17	0	1	0	9	2	5

Thus, *the horizontal QRS loop in chronic cor pulmonale* and, to a lesser extent, in chronic emphysema *displayed two maximal mean instantaneous QRS vectors*. The first of these had an average orientation varying between −30 and −65, while the second maximal vector was located on the average at about −105 (Fig. 133). Although the magnitudes of the two mean instantaneous vectors just referred to were often approximately equal, not infrequently in the chronic cor pulmonale cases the second instantaneous vector was the larger of the two.

Right sagittal QRS loop.—The sagittal QRS loop in the type C pattern resembled that already described in the type A pattern with the exception that, by and large, the terminal mean instantaneous QRS vectors were situated superiorly less frequently than in the latter pattern. This difference was especially prominent in the vectorcardiograms of the mitral stenosis patients.

Frontal QRS loop.—Like the sagittal loop, the frontal QRS loop in the type C pattern also tends to

the loop as a whole—may well reflect clockwise rotation of the heart on its longitudinal axis plus some degree of right ventricular preponderance. On the other hand, the type C QRS sÊ loop pattern in the vectorcardiograms of our cases of mitral stenosis would seem to represent primarily the electrical effects of anatomic cardiac rotation. Obviously, these conclusions are only tentative, and undoubtedly they will be altered as further information becomes available.

As one might anticipate, the electrocardiographic findings in mitral stenosis with the type C QRS sÊ loop pattern consist for the most part *of right axis deviation* of A QRS in the bipolar limb leads and the precordial lead QRS pattern of moderate or marked clockwise rotation. In general, the electrocardiographic *findings in the chronic cor pulmonale* and chronic emphysema cases with the type C vectorcardiographic pattern were much the same as those just described, except that the right axis deviation and clockwise rotation were generally of more

marked degree than customarily observed in mitral stenosis

Genesis of Vectorcardiographic QRS sE Loop Patterns

In chronic cor pulmonale just as in mitral stenosis the earliest and most marked evidence of anatomic right ventricular hypertrophy consists of thickening of the trabecular and papillary muscles and the muscle around the base of the outflow tract of the right ven [illegible] The [illegible] from [illegible] *the posterior base* [illegible] in fundibulum of the right ventricle to the right atrial ostium According to Sodi Pallares the crista supraventricularis and adjacent regions of the outflow tract of the right ventricle belong to the interventricular septum at its highest level and together with basal right septal myocardium are normally the last portion of the right ventricle and one of the last regions of the heart to complete depolarization

The QRS forces produced in the basal right ventricular myocardium are directed *superiorly* to the right, and somewhat anteriorly but algebraic summation of these forces with the larger *leftward* posterior and inferior or superior forces arising in the basal left ventricular wall normally yields *terminal* resultant or mean QRS vector directed posteriorly somewhat to the left and either superiorly and inferiorly However because of the physiologic right ventricular preponderance existing in young children the larger basal right ventricular *forces* may produce terminal mean QRS vectors directed to the right, superiorly and posteriorly If these vectors are situated within the 160 *to* −150 segment *of* the horizontal reference frame they *project* small terminal R deflections on lead V_1 *and* V_{3R} Since the component forces and the resultant terminal vectors *are* not normally of great magnitude the R deflections appearing in these instances in the right precordial leads are usually quite small rarely exceeding 4 mm

Inasmuch as any terminal *mean instantaneous* QRS spatial vector is the resultant of component left ventricular forces directed to the left posteriorly and superiorly or inferiorly and of componen [illegible] [illegible] magnitude (assuming for simplicity that their direction remains unchanged which is unlikely *to be the case in* right ventricular hypertrophy) the *resultant* or *mean instantaneous* QRS vectors *must* necessarily rotate in a counterclockwise direction from left posterior and superior or inferior to right posterior and superior and eventually to right anterior and superior To a certain extent this hypothetical sequence of changing orientation of the terminal QRS vectors is observed clinically in the electrocardiogram and vectorcardiogram but certain discrepancies between fact and theory *are noted* particularly in chronic cor pulmonale For example the *anticipated rightward superior* and anterior terminal QRS vectors *reflecting* selective *hypertrophy of the right ventricle* are rarely found in chronic cor pulmonale and pulmonary emphysema instead in these conditions the *terminal* QRS vectors are generally *directed* to the right posteriorly and superiorly or slightly to the left posteriorly and inferiorly This may be due in no small part to the associated changes in heart position and cardiac *rotation* which are known to occur in emphysema As previously indicated the lowering of the diaphragm as the result of emphysema in turn causes the heart to descend in the chest to assume a more vertical position and to rotate in a clockwise direction around its longitudinal axis

In the past it was accepted without much question that the electrocardiographic effects of cardiac positional and rotational changes in emphysema simply reflected orientation of the *exploring* lead electrode *to an* aspect of the heart different from normal However this explanation is based on the premise that the electrocardiographic leads respond *primarily* to "proximity potentials" produced by the region of the heart closest to the exploring electrode and *as pointed out in Chapter 4 this concept is* probably not valid when applied to body surface leads In all likelihood the correct explanation of the electrical effects of variations in anatomic heart position and rotation such as those occurring in chronic emphysema is to be sought *in* studies of the *effects of these* factors on the direction and magnitude of the lead vectors or *effective axes For the present* one can only *speculate as to the manner* in which positional and rotational *changes of the heart may possibly be* related to the terminal *rightward* superior and posterior QRS forces *so* frequently observed in chronic *cor pulmonale In the following* paragraphs *there is presented a purely* hypothetical mechanism by which the *changes in heart position* and rotation in *chronic cor pulmonale may* account *for* the characteristic *rightward* posterior and superior terminal QRS forces

As we indicated in the introduction to this *section* of the text (Chapter 13) the changes in heart position and rotation in pulmonary emphysema must

stantaneous vector was simply rotated abnormally far *posteriorly its average orientation being about −40* In contrast the horizontal QRS loop in the chronic cor pulmonale and chronic emphysema cases displayed the following features

1 The initial deflection of the loop was written almost straight anteriorly *or sometimes slightly to the* right or left and anteriorly

2 The loop then turned abruptly posteriorly and *the efferent limb of the loop* was written as an almost straight line far posteriorly and to a varying degree to the left

3 On reaching its maximal leftward and posterior extent the loop then turned *medially and proceeded* rapidly from left to right

4 The afferent limb and terminal part of the loop were often written in the form of a large rightward and posterior deflection much like that occurring in the fully developed type A QRS sÊ loop pattern

resemble that occurring in the type A pattern although generally being oriented somewhat more inferiorly If the frontal QRS loop should happen to be oriented almost vertically inferiorly then the horizontal QRS loop may simulate that observed in the type A QRS sÊ loop pattern however this is simply the result of foreshortening of the loop in the horizontal projection (Fig 134 and Table 16)

We are not certain as to the manner of production or the *significance of the type C QRS sÊ loop pattern* although we have observed 2 patients with chronic cor pulmonale with this pattern who were found at postmortem to have marked right ventricular hypertrophy Although *there is no other evidence to support* this impression we nonetheless feel that the type C pattern as it appears in the chronic cor pulmonale cases—that is with large rightward posterior and *superior terminal mean instantaneous QRS vectors* in addition to the abnormal posterior rotation of

TABLE 16 —QRS sÊ Loop Patterns in Mitral Stenosis Chronic Cor Pulmonale and Chronic Pulmonary Disease without Cor Pulmonale

	Total No of Cases	Normal QRS sÊ	Right Ventricular Hypertrophy	Left Ventricular Hypertrophy	Type A QRS sÊ	Type B QRS sÊ	Type C QRS sÊ
Mitral stenosis	54	3	32	4	9	3	3
Chronic cor pulmonale	20	0	3	0	5	6	6
Chronic pulmonary disease	17	0	1	0	9	2	5

Thus the horizontal QRS loop in chronic cor pulmonale and to a lesser extent in chronic emphysema displayed two maximal mean instantaneous QRS vectors The first of these had an average orientation varying between −30 and −65 while the second maximal vector was located on the average at about −105° (Fig 133) Although the magnitudes of the two mean instantaneous vectors just referred to were *often approximately equal not infrequently* in the chronic cor pulmonale cases the second instantaneous vector was the larger of the two

Right sagittal *QRS loop —The sagittal QRS loop* in the type C pattern resembled that already described in the type A pattern with the exception that by and large the terminal mean instantaneous QRS vectors were situated superiorly less frequently than in the latter pattern This difference was especially prominent in the vectorcardiograms of the mitral ste*nosis patients*

Frontal QRS loop —Like the sagittal loop the frontal QRS loop in the type C pattern also tends to

the loop as a whole—may well reflect clockwise rotation of the heart on its longitudinal axis plus some degree of right ventricular preponderance On the other hand the type C QRS sÊ loop pattern in the vectorcardiograms of our cases of mitral stenosis would seem to represent primarily the electrical effects of anatomic cardiac rotation Obviously these conclusions are only tentative and undoubtedly they will be altered as further information becomes available

As one might anticipate the electrocardiographic *findings in mitral stenosis* with the type C QRS sÊ loop pattern consist for the most part of right axis deviation of A QRS in the bipolar limb leads and the precordial lead QRS pattern of moderate or marked clockwise rotation In general the electrocardiographic findings in the chronic cor pulmonale *and chronic emphysema cases with the type C vector*cardiographic pattern were much the same as those just described except that the right axis deviation and *clockwise rotation were generally of* more

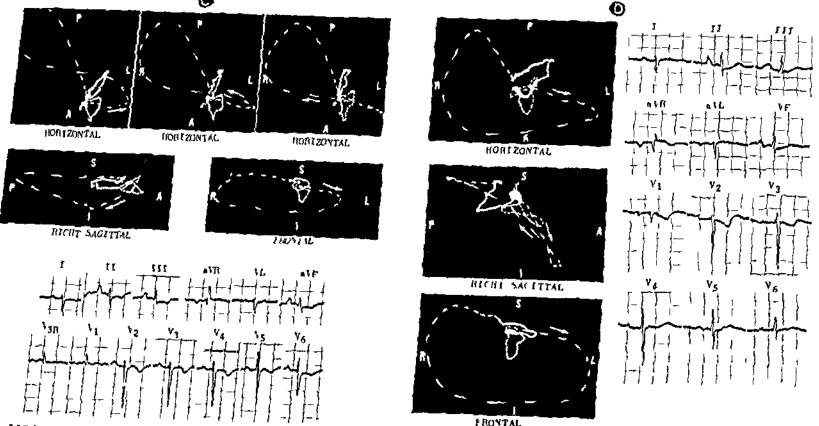


Fig 135 (*cont.*)—Four months later (**C**) the electrocardiogram showed prominent P waves in leads II, III and aVF, right axis deviation of Â QRS (−1?0°), low amplitude qRS deflections in leads V3R and V1, orientation of Â QRS in the horizontal plane to the right and posteriorly, and inverted T waves in leads II, III, aVF and V1 through V6. *The authors interpret this electrocardiogram as showing right ventricular hypertrophy and anterior myocardial ischemia and/or digitalis effect.* In the vectorcardiogram in **C** the configuration of the horizontal QRS loop resembles that in **B** except that with respiration the left to right limb of the loop tended to shift progressively farther anteriorly while the magnitude of the terminal right posterior mean instantaneous vectors far exceeded that of earlier leftward vectors. Thus the greater part of the horizontal and frontal QRS loops was written to the right of the midline. The anterior and vertically inferior orientation of the P sÊ loop and the increased magnitude of the instantaneous P vectors are compatible with P pulmonale while the posterior orientation of the T sÊ loop is consistent with anterior myocardial ischemia. Again the vectorcardiographic findings in **C** would be interpreted by the authors as being indicative of right ventricular hypertrophy. The electrocardiogram and vectorcardiogram in **D** were recorded about 8 months after those in **C** and about 2 months prior to the patient's death. *The electrocardiogram is less suggestive of right ventricular hypertrophy than that in* **C**. There is right axis deviation of Â QRS; however, lead V1 displays an essentially downwardly directed QRS deflection. On the other hand, the vectorcardiogram displays the features of P pulmonale and quite typical findings of right ventricular hypertrophy. The horizontal QRS loop is entirely clockwise inscribed and is situated anteriorly except for the terminal portion of the loop. One gets the impression that the principal difference in QRS loop configuration between the vectorcardiogram in **C** and that in **D** is that the left to right limb of the QRS sF loop in **D** is displaced far anteriorly. In fact, if one compares the planar QRS loops in records **B–D**, it becomes apparent that they differ only to the extent that there is progressive anterior displacement of the efferent left-to-right limb of the QRS sF loop culminating with the fully developed right ventricular hypertrophy pattern in **D**.

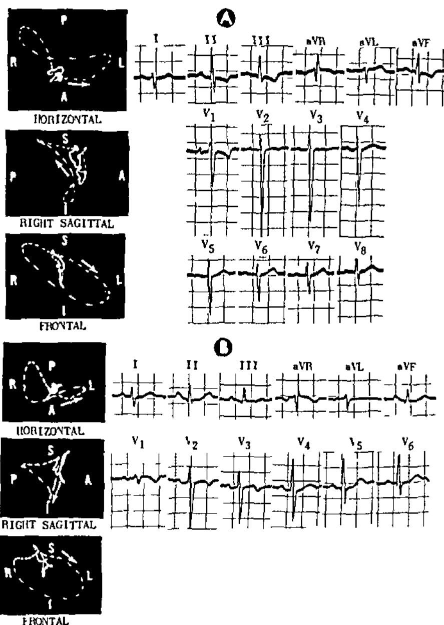


Fig 135 —A electrocardiographic and vectorcardiographic patterns of chronic cor pulmonale recorded from a man 73 with congestive heart failure and pulmonary insufficiency due to bilateral far advanced tuberculosis and bullous pulmonary emphysema Th...
in the horizontal plane -
pulmonary disease In th
horizont il QRS loop has
left and posteriorly and the second half inscribed clockwise equally far to the right and posteriorly The terminal portions of the right sagittal and frontal QRS loops are located superiorly to the right and posteriorly In the authors opinion ... pulmonale
B ... 75 for whom the postmortem findings (he
di ... physema advanced saccular bronchiectasis of
the right middle and lower lobe ...
right ventricular and advance
corded about 1 year before hi
strable right ventricular hype
minor degree of right axis dev
which is considered by the au

changes in the QRS sE loop On the other hand one can easily conceive of respiration producing instant to instant alteration in the direction of the effective axes of the vectorcardiographic leads by virtue of small shifts in the location of the equivalent cardiac dipole changes in conductivity of the lungs etc

3 From a patient with severe chronic cor pulmonale we recorded serial electrocardiograms and vectorcardiograms at 2–4 month intervals over a period of about 1½ years the last records being made shortly before the patient s death Postmortem examination confirmed the presence of marked anatomic right ventricular hypertrophy Of the many electrocardiograms obtained during the time the patient was followed clinically there were only two which showed diagnostic evidence of right ventricular hypertrophy and these were recorded quite late in the course of the patient s illness (Fig 135) The initial vectorcardiogram displayed the type B QRS sE loop pattern As the patient s condition (chronic cor pulmonale) worsened clinically the efferent left to-right limb of the QRS sE loop which was at first posteriorly situated shifted steadily forward until finally it reached a point far enough anterior to cause a complete reversal in the direction of inscription of the horizontal QRS loop (the entire loop having a clockwise direction of inscription) and to produce a figure-of eight configuration of the sagittal QRS loop Despite the

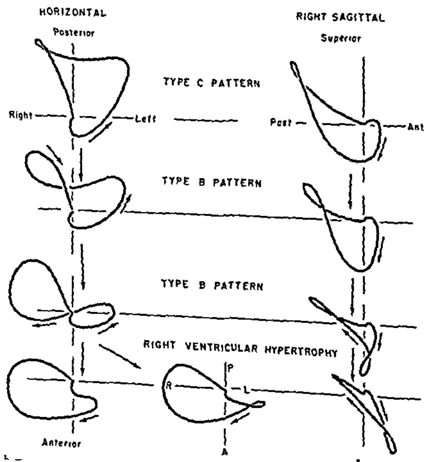


Fig 136 —
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type

necessarily be accompanied by a shift in the location of the cardiac dipole center The altered location of the dipole plus the decreased conductivity of the emphysematous lungs in turn lead to changes in the effective axes of the various leads However of these two factors the more important is the location of the cardiac dipole Schaffer has demonstrated that with a shift in the location of the dipole center toward a given lead electrode the image point of the electrode is shifted farther away from the dipole on the other hand if the dipole moves away from the electrode the image point of the latter shifts closer to the dipole Presumably as the heart descends to a lower level in the chest in pulmonary emphysema the dipole center also descends In so doing it approaches more closely the anterior electrode of the sagittal vector cardiographic lead lead Z and therefore causes the effective axis of lead Z to rotate clockwise If this supposition is correct then it may well be that in chronic cor pulmonale (due to emphysema) the normal downward slant of lead Z in a posteroanterior direction is exaggerated This being the case the manifest vectors are recorded by lead Z as if rotated counterclockwise vertically inferior vectors being rotated in an anterior direction and vertically superior vectors in a posterior direction This effect of the increased downward slant of lead Z is all the more marked the more nearly perpendicular the cardiac vectors lie to the anatomic axis of lead Z Thus in chronic cor pulmonale with selective hypertrophy of the basal right ventricle the late rightward anteriorly and superiorly directed QRS forces of increased magnitude actually are recorded as if oriented posteriorly because of the marked downward tilt of the effective axis of lead Z on the other hand similarly directed QRS forces are more likely to be recorded in their true anterior orientation in mitral stenosis or congenital heart disease because of the less marked rotation of the effective axis of lead Z in these conditions

In a small number of cases of chronic cor pulmonale with vectorcardiographic type A type B or type C QRS sÊ loop patterns we tested the above premise in the following manner After the control vectorcardiogram had been recorded in the customary way a second record was obtained with the anterior electrode of lead Z applied at a higher level on the chest This was done in an attempt to reverse the postulated exaggerated posteroanterior downward slant of lead Z produced by pulmonary emphysema On comparing the two vectorcardiograms recorded in each of the cases of chronic cor pulmonale studied it was found that in the second vectorcardiogram the terminal portion of the QRS sÊ loop originally located to the right superiorly and posteriorly was found to lie anteriorly It is highly probable that the deviation of the superiorly oriented vectors from posterior to anterior in the second vectorcardiogram reflected the upward slant of lead Z in a posteroanterior direction

While the above observations do not prove they at least suggest the following (*a*) that the mechanism by which cardiac positional and rotational changes in pulmonary emphysema influence the electrocardiogram and vectorcardiogram entails some alteration in the magnitude and especially in the direction of the effective axes of the leads utilized and (*b*) that the unexpected posterior orientation of the terminal QRS forces in chronic cor pulmonale representing for the most part the activation forces arising in the hypertrophied basal region of the right ventricle may be related etiologically to clockwise rotation of the effective axis of lead Z There are certain additional reasons for believing that the terminal right posterior and superior QRS forces in chronic cor pulmonale reflect selective right ventricular hypertrophy some of these are as follows

1 In almost all of the vectorcardiograms diagnostic of right ventricular hypertrophy with rightward and posterior terminal QRS vectors in our cases of mitral stenosis chronic cor pulmonale and congenital heart disease as well as in the great majority of similar vectorcardiograms published in Grishman and Scherlis textbook of vectorcardiography and in most of the vectorcardiograms of this type described in the report of Richman and Wolff the terminal vectors were also oriented inferiorly However the converse of this did not hold true—that is superiorly located terminal QRS vectors were by no means invariably directed posteriorly Nevertheless the consistency of the former association would seem to imply some relationship between the posterior orientation of the terminal QRS vectors in chronic cor pulmonale and the fact that these vectors are situated superiorly

2 In a number of mitral stenosis cases we have noted respiratory variation in QRS sÊ loop configuration between a typical RSR pattern of right ventricular hypertrophy and an equally typical type B QRS sÊ loop pattern Generally this was accompanied by a changing orientation of the terminal QRS vectors from slightly inferior to slightly superior Such a fleeting transition back and forth between the foregoing two QRS sÊ loop patterns can hardly be attributed to such a relatively permanent characteristic as the degree of anatomic right ventricular hypertrophy nor would it seem likely that respiration alone could cause beat to beat variation in heart position and/or rotation of sufficient degree to produce the observed

seems reasonably certain that many instances of incomplete right bundle branch block pattern found in pulmonary embolism are not actually right bundle branch block at all—at least not in the sense that the term is used elsewhere in this text (see Chapter 17) As will be explained later in the description of the vectorcardiographic features of pulmonary embolism (pp 219 and 220) in many of the cases in which a *terminal R wave is recorded in lead* V_1 *of the electrocardiogram* the configuration of the QRS sÊ loop will not resemble right bundle branch block at all Whether the electrocardiographic and vectorcardiographic abnormalities in these cases result from a local or focal intraventricular block secondary to marked localized dilatation of the pulmonary outflow tract or are produced by some other mechanism remains to be determined

Acute coronary insufficiency —As a result possibly of a fall in cardiac output myocardial anoxemia may occur particularly in older subjects and those patients with pre-existing coronary artery disease and left ventricular enlargement Usually elevated sometimes depressed S–T segments and/or inverted T waves are recorded in leads V_1 and V_2 and depressed S–T segments and upright T waves appear in leads V_5 and V_6

Abnormalities probably related primarily to cardiac rotation *Right-axis deviation of Â QRS and the* S_I–Q_{III} *pattern of McGinn and White* —In all probability anatomic cardiac rotation occurs in acute pulmonary embolism as the result of right ventricular and atrial dilatation However it is not certain whether rotation plays a major or a minor role in the etiology of the right axis deviation of the instantaneous QRS vectors in the frontal plane and their posterior rotation in the horizontal plane In any event with moderate degrees of right axis deviation deep *terminal S* waves are projected on lead I *and prominent* Q waves of less than 0 03 second *duration on* leads III and aVF This finding plus depressed S–T segments with "*staircase ascent*" *to the* T wave in lead I constitutes the S_I–Q_{III} pattern of McGinn and White which is considered quite characteristic of pulmonary embolism *If there is a marked degree of*

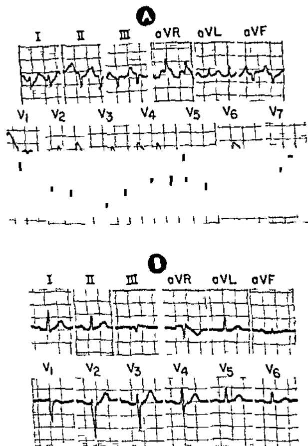


Fig 137 —Electrocardiograms recorded from a woman, 23 (A) 3 days post partum shortly after clinical onset of acute cor pulmonale due to pulmonary embolism and (B) 1 day later The findings in A quite typical of acute cor pulmonale are marked right axis deviation of Â QRS producing an S_I–S_{II}–S_{III} pattern I pulmonale pattern in the limb leads QS deflections in leads III and aVF precordial leads showing the QRS pattern of "marked clockwise rotation" with low R waves and relatively deep S waves in all leads from V_1 through V_7 and S–T segment depression in leads I II and V through V The heart rate in A is approximately 180 beats per minute and the rhythm is probably paroxysmal atrial tachycardia Electrocardiogram B is within normal limits the foregoing findings having disappeared

appearance of typical features of right ventricular hypertrophy the QRS sÊ loop continued to display terminal QRS vectors directed to the right posteriorly and superiorly Obviously the patient s anatomic right ventricular hypertrophy did not have its onset only during the final year and a half of life but must have long antedated the first vectorcardiogram recorded

This finding and the close similarity of the QRS sÊ loop configuration in records displaying the type B pattern and those with the pattern of vectorcardiographic right ventricular hypertrophy would seem to suggest that the type B QRS sÊ loop pattern in some cases at least constitutes a third vectorcardiographic *pattern of right ventricular hypertrophy modified* however by the distorted recording response of the vectorcardiographic leads In fact this opinion as to the significance of the type B QRS sÊ loop pattern does not originate with us but has been expressed by Fowler and Helm Grishman and his associates and others although this same pattern has been considered a normal variant by other investigators It must be admitted that we have on rare occasion observed a type B QRS sÊ loop pattern in vectorcardiograms of normal subjects but for that matter the same holds true for the RSR pattern of right ventricular hypertrophy Consequently we do not feel that these rare exceptions to the rule can be regarded as evidence militating against the possibility that the type B pattern is indicative of right ventricular hypertrophy As a matter of fact it may well be that the type A and type C QRS sÊ loop patterns also signify either lesser degrees of anatomic right ventricular hypertrophy or right ventricular hypertrophy plus rotational and positional changes of the heart The authors have observed the transition of a type C QRS sÊ loop pattern to a type B pattern as a respiratory variation in occasional patients and have also followed the evolution of a type B QRS sÊ loop pattern into a typical right ventricular hypertrophy pattern in other cases (Fig 136) From a purely practical standpoint the type B pattern in particular has the significance that if it is eventually proved with certainty that the type B pattern is indicative of right ventricular hypertrophy then the physician will be able to recognize right ventricular hypertrophy in a significant number of patients with chronic cor pulmonale whose electrocardiograms provide no clue as to the diagnosis

ACUTE PULMONARY EMBOLISM WITH ACUTE COR PULMONALE ECG AND VCG FINDINGS

When pulmonary embolism is suspected clinically one of the most important decisions which must be made promptly is whether or not the clinical picture although resembling pulmonary embolism is actually due to myocardial infarction For the electrocardiographer the exclusion of myocardial infarction takes precedence over the diagnosis of pulmonary embolism The electrocardiographic differentiation of infarction and pulmonary embolism ordinarily does not present too great a problem but sometimes the distinction between the two can be quite difficult to make

The most characteristic feature of the electrocardiogram in pulmonary embolism—the precipitant appearance and usually transient duration of the electrocardiographic changes—is also the chief reason why this condition has been so difficult to study adequately clinically Nevertheless it has been shown that one can produce in dogs by compression of the pulmonary artery or by experimental pulmonary embolism the same electrocardiographic abnormalities as those observed in patients with pulmonary embolism In clinical pulmonary embolism the electrocardiogram may show no changes whatsoever or it may display one or more of the following abnormalities

TRANSIENT ATRIAL OR SUPRAVENTRICULAR ARRHYTHMIAS —These arrhythmias may take the form of premature atrial extrasystoles paroxysmal atrial or nodal tachycardia atrial fibrillation or atrial flutter

P PULMONALE —The P pulmonale pattern of the P waves is not so common a finding in pulmonary embolism as was once thought As will be recalled P pulmonale can be diagnosed when tall P waves ($\geqq$2 5 mm) are present in leads II III and aVF and low upright P waves in lead I Presumably pulmonary embolism causes dilatation of the right atrium which brings the wall of the atrium closer to the chest wall thereby increasing the voltage recorded by the lead electrodes In addition right atrial dilatation also causes the mean instantaneous P vectors to rotate inferiorly to a more vertical position

TRANSIENT COMPLETE OR INCOMPLETE RIGHT BUNDLE BRANCH BLOCK —Right bundle branch block occurring with pulmonary embolism is probably due to injury sustained by *the long trunk of the right bundle* branch secondary to right ventricular dilatation It

right axis deviation the instantaneous QRS vectors may come to lie in the right upper quadrant of the frontal reference frame so that an S_I–S_{II}–S_{III} pattern may appear in the standard bipolar limb leads while the remaining extremity leads record rS deflections with the exception of lead aVR which registers a Qr or QR deflection

Pattern of "marked clockwise rotation" in the precordial leads—One of the commonest electrocardiographic findings in pulmonary embolism or acute cor pulmonale consists of a shift of the QRS transition point or lead (the lead registering an equiphasic RS deflection) farther to the left, sometimes beyond lead V_6 This is simply an expression of the fact that the mean QRS vector in the horizontal plane is rotated posteriorly ...

Qr qr or QS deflection The S–T segments in the right precordial leads may be either depressed or elevated while the T waves are usually inverted in these leads Grishman attributes the T wave inversion and the apparent subepicardial injury pattern in the right precordial leads to rotation of the T sE loop and the subendocardial injury vector along with the QRS sE loop Consequently inverted T waves and elevated S–T segments are projected on right precordial leads and on leads III and aVF and depressed S–T segments and upright T waves are projected on left precordial leads On the other hand Sodi Pallares attributes the S–T segment elevation and T wave inversion in right precordial leads to the effects of injured areas which may be present either on the right septal surface or on the free wall of the right ventricle As a general rule the S–T segment and T wave changes accompanying pulmonary embolism last only a few days although they may be of even briefer duration while the rotational QRS changes can persist for 1–3 weeks after the acute episode (Figs 137–139)

Although a few investigators have managed to accumulate sizable series of cases of pulmonary embolism and have reported their vectorcardiographic observations by and large the general experience in the vectorcardiographic diagnosis of this condition is still somewhat limited The reasons for this dearth of reported studies are multiple—for example the abrupt onset and evanescence of the clinical manifestations of acute cor pulmonale due to pulmonary embolism

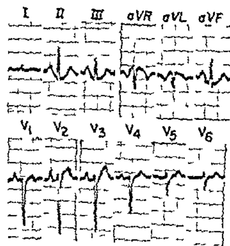
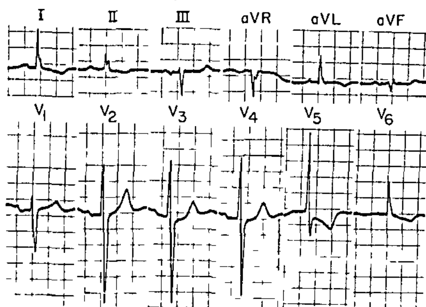


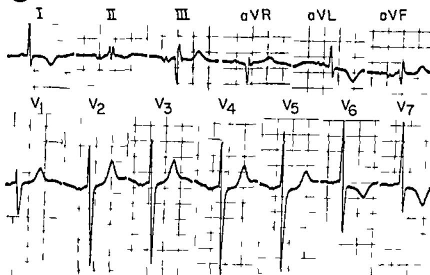
Fig 139 —Electrocardiogram in acute pulmonary embolism recorded shortly after onset of the embolism The findings are ...

the critical state of the patient and the relative immobility of the cumbersome types of ...

... graphically 15 cases of pulmonary embolism In some instances vectorcardiograms were available which had been recorded before the onset of the pulmonary embolism The major vectorcardiographic findings according to these investigators were

Fig 138 —Serial electrocardiograms recorded ... embolism ... leads ... mately ...





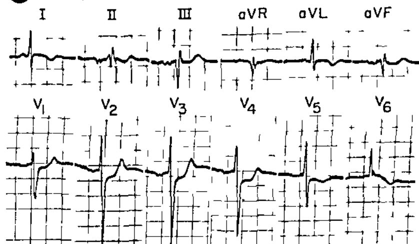


FIG 138 —(*Legend on facing page*)

Left Bundle Branch Block

GENERAL CONSIDERATIONS

Most of what is known about the manner in which the septum and ventricles undergo activation in bundle branch block has been derived from studies of induced block in the dog heart but how closely experimental block in the dog heart duplicates clinical block in the human heart is at present a moot question In fact there is growing suspicion that significant differences exist between experimental and clinical bundle branch block but more will be said of this later during the discussion of right bundle branch block (Chapter 17) Even if one assumes the essential similarity of the clinical and experimental lesions bundle branch block still retains most of it controversial aspects since the observations made in experimental block have been interpreted differently by different authorities. To these [illegible]

[illegible] branch block will be deferred for individual discussion along with discussion of other related aspects of each type of bundle branch block

The normal intraventricular conducting pathways In the normal heart the bundle of His bifurcates [illegible]

[illegible] and branches profusely thereafter In the dog heart almost the entire thickness of the septal muscle mass is contributed by the left ventricle and normally is activated via the left bundle branch and its ramifications By way of contrast the right bundle branch is a long slender nonbranching stalk through most of its course down the right septal surface Its first ramifications appear in the vicinity of the base of the right anterior papillary muscle—that is on the endocardial surface of the anterior wall of the right ventricle near the interventricular septum From this region excitation spreads upward over the right septal surface to activate the underlying thin layer of muscle in an apex to-base direction

The significance of the different anatomy and distribution of the two bundle branches lies in the fact that the slender cross sectional dimension of the right bundle branch and its longer course prior to branching render it far more vulnerable to small focal fibrotic degenerative or inflammatory lesions than the freely branching left bundle branch This being the case the incidence of right bundle branch block would exceed that of left bundle branch block were it not for the fact that the pathologic processes most likely to produce bundle branch block involve the left ventricle more often than the right Thus one predisposing factor tends to counterbalance the other and on the average the electrocardiographic patterns of left bundle branch block and right bundle branch block are noted clinically with about equal frequency

In clinical [illegible]

[illegible] QRS intervals of less than 0.12 but greater than 0.08 second *incomplete* [illegible]

[illegible] *complete* " Incomplete left bundle branch block will always be so designated and is discussed separately later in this chapter

Diffuse intraventricular block will not be discussed in any great detail in this text since it is sufficient to state that this type of conduction defect is characterized by widening of the QRS complexes often with relatively little change in their configuration Diffuse intraventricular block can occur spontaneously but is more often a manifestation of severe myocardial damage or of excess of quinidine or of pronestyl effect or of hyperkalemia

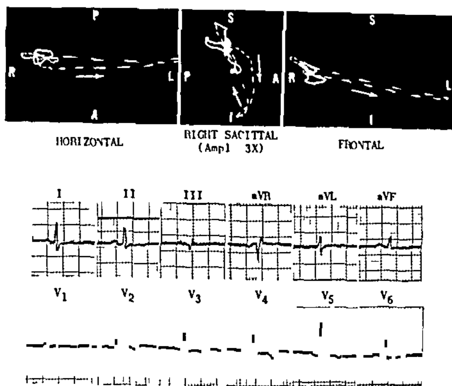


Fig 140 —Electrocardiogram and vectorcardiogram recorded about 24 hours after clinical onset of acute pulmonary embolism *The electrocardiogram* displays an S_I–Q_{III} pattern of minor prominence and inverted T waves in leads V_1 through V_4 with flat or diphasic T waves in leads I and V_6 These findings are not sufficient to justify an unequivocal di am features are the anterior displacement of the QRS sÊ rected to the right posteriorly and superiorly These vec Karlen and Wolff in a number of cases of acute pulmonary embolism

1 There is transient clockwise rotation of the QRS sÊ loop around its longitudinal axis This is evidenced in the vectorcardiogram by the more superior and less rightward orientation of the initial vectors of the QRS sÊ loop Occasionally the initial vectors are directed to the left

2 The sagittal QRS loop is sometimes inscribed in a counterclockwise direction although clockwise inscription occurs more often in pulmonary embolism

3 The frontal QRS loop usually has a clockwise direction of inscription The terminal instantaneous vectors of the frontal QRS loop are almost invariably oriented to the right

4 When the initial deflection of the QRS sÊ loop is directed superiorly in pulmonary embolism the magnitude and duration of the instantaneous vectors of this deflection are greater than normal but less than in diaphragmatic myocardial infarction

5 The terminal instantaneous vectors of the QRS sÊ loop are almost always situated to the right and posteriorly and as often as not lie superiorly Moreover the magnitude of these vectors also exceeds normal limits (Fig 140)

We have obtained vectorcardiograms in a few cases of pulmonary embolism but our experience is too limited to permit any conclusions concerning either the diagnostic findings or the usefulness of the vectorcardiogram in this condition On the whole the vectorcardiographic abnormalities in our cases were similar to those described by Karlen and Wolff The most consistent vectorcardiographic finding in our cases was the presence of a terminal deflection of the QRS sÊ loop to the right posteriorly and superiorly much like that observed in the types A and B loop patterns in mitral stenosis and chronic cor pulmonale (previously described) However the terminal mean instantaneous QRS spatial vectors in our cases of acute cor pulmonale never attained a magnitude comparable to that of the terminal vectors in the types A and B QRS sÊ loop patterns

left bundle branch block and probably reflects the fact that both component vectors are exerting a significant effect on the direction of the resultant vector As a consequence the 0 01 second VA vector is situated at some point intermediate between the rightward and anteriorly directed right ventricular vector and the leftward and posteriorly directed septal vector so that the VA vector generally is oriented slightly anteriorly and to the left The components of the

Leads I and V_6 —Beginning upstroke of a wide slurred or notched R wave

Lead aVF —Upstroke of a small R wave

Lead V —Small initial R wave The peak of this R wave in lead V_1 coincides with the onset of the intrinsicoid deflection and occurs at the normal time in left bundle branch block (normal 0 015–0 035 second)

POSTERIOR LEFTWARD AND INFERIOR ORIENTATION —*This orientation of the initial VA vector is observed somewhat less frequently than that described above* Presumably the leftward and posteriorly directed septal component vector *dominates* the electrical field of the heart *from the very* onset of the QRS interval Whether the electrical predominance of the early leftward and posterior forces is due to their greater magnitude or to a delayed or slower development of the rightward and anterior forces of right ventricular activation is not known at present

Leads I and V —Beginning upstroke of a wide slurred or notched R wave

Lead aVF —Upstroke of a small R wave

Lead V_1 —Beginning downstroke of a broad deep QS deflection

0 02 0 04 AND 0 06-SECOND VA VECTORS

From about 0 02 to 0 06 second after onset of ventricular excitation depolarization of the muscle mass of the septum continues in a right-to-left direction Normally the septal musculature is activated so rapidly via the Purkinje fibers that the potentials generated are relatively negligible however in left bundle branch block septal excitation probably occurs in an aberrant manner so that the resulting *forces* have not only a different direction but also a greater magnitude Thus the 0 02 0 04 and 0 06-second VA vectors appearing during this period increase in magnitude as they develop to the left posteriorly and superiorly They tend to be located on the average between −30 and −80 in the horizontal plane and between +10 and −10 in the frontal plane If one were to imagine these three vectors to be projected on the horizontal plane they would be *observed to develop in a clockwise direction*

Leads I and V_6 —The major part of the upstroke of the R wave

Leads aVF and V_1 —The major part of the downstroke of the QS or rS deflection recorded in these leads

0 08 SECOND VA VECTOR

The VA vector appearing at about 0 08 second after onset of the QRS interval is the largest instantaneous vector produced and is directed to the left and more superiorly and posteriorly than the other

to fiber through the left ventricular myocardium along a broad activation front The electrical forces produced are therefore of increased magnitude As was pointed out in an earlier chapter the Purkinje fibers penetrate deeply into the subendocardium so that normally with the onset of ventricular excitation

activation wave spreads concentrically from each of these islands with the result that activation of the inner muscle layers normally occurs in too many directions to give rise to measurable electrical forces Once the islands of negativity merge to form a bounded wave front measurable forces appear Grant and Dodge speculate that in left bundle branch block left ventricular excitation may no longer begin as small islands of negativity but may begin as a wave front spreading from the right ventricle This would account at least in part for the increased magnitude of the electrical forces in left bundle branch block and therefore for the wide R waves recorded in leads I aVL and V_6 and the deep broad S waves registered in the right precordial leads

2 There is reason to believe that in left bundle branch block in the human heart the posterobasal wall of the left ventricle is activated before the anterolateral wall Thus the thickest

the poster

at the peak of the R wave or with the end of the slurred plateau like peak of

MECHANISMS OF LEFT BUNDLE BRANCH BLOCK

The salient characteristic of left bundle branch block which differentiates it from other disturbances of intraventricular conduction is that septal and left ventricular activation from onset to termination is altered by the block The manner in which excitation spreads through the septum and ventricles in left *bundle branch block may be outlined as follows*

1 Since conduction down the left bundle branch is *blocked at a high level in the conducting pathway* the excitation impulse first appears low on the right septal surface near the base of the anterior papillary muscle
2 From this point excitation spreads over the endo*cardial surface of the right ventricle* and upward over the right septal surface
3 As the activation wave moves transmurally through the apicolateral free wall of the right ventricle the muscle mass of the septum undergoes activation *in a right to left and apex to base direction* The excitation wave proceeds so slowly through the left septal muscle mass as to suggest that it spreads from muscle fiber to muscle fiber rather than via the specific conducting fibers of the Purkinje system
4 The explanation for the QRS prolongation in left bundle branch block and the manner in which the wave of excitation spreads through the free wall of the left ventricle are the source of considerable controversy There are perhaps three main schools of thought
   a) It is widely believed that the septum is the region of conduction delay excitation spreading either slowly over normal pathways or at a normal rate over abnormal and longer pathways Once the excitation impulse reaches the left septal surface it enters *the left bundle* branch below the block and is transmitted over normal pathways to the left ventricle
   b) Rodriguez and Sodi Pallares are in agreement with the above version with this exception they believe that the main conduction delay in left bundle branch block is localized to a specific region of the septum namely the transi*tion zone between regions of septum excited* via the right branch and those excited via the left branch Once the excitation impulse jumps this barrier and reaches the ramifications of the left bundle branch it spreads over the left septal surface and through the left ventricle in a normal manner
   c) The third school of thought which seems to be gaining increasing support holds that there *is* fiber to fiber transmyocardial spread of excitation not only through the septum but also through the left ventricle Grant and Dodge Kennamer and Prinzmetal and Wener and his *associates among others have produced* evidence in support of this contention The last named investigators used esophageal and precordial leads to demonstrate that in clinical left bundle branch block the posterobasal wall of the left ventricle consistently undergoes activation from 0 01 to 0 08 second earlier than the anterolateral wall—just the reverse of the *normal sequence*

THE INSTANTANEOUS VA VECTORS

The spread of activation through the interventricular septum and ventricles in left bundle branch block and the electrical forces produced can be presented in simplified manner in terms of the instantaneous VA vectors (see also Fig 141)

0 01 SECOND VA VECTOR

In left bundle branch block the 0 01 second VA vector may be considered the resultant of two component vectors (*a*) a *septal vector* directed to the left posteriorly and inferiorly which represents the electrical *forces produced by early activation of the sep*tum in a right to left and apex to base direction and (*b*) a *right ventricular vector* directed anteriorly somewhat to the right and inferiorly which represents the electrical forces generated during simultaneous activation of the apicoanteromedial wall of the right ventricle Depending in all probability on the *exact time sequence of excitation* of the right septal surface and the apical right ventricular muscle and on the relative magnitudes and directions of the component electrical forces arising *in these two re*gions the resultant 0 01 second VA vector may be oriented in either of two directions (1) anterior leftward and inferior or (2) posterior leftward and inferior

ANTERIOR LEFTWARD AND INFERIOR ORIENTATION —This is the more common of the two general directions which the 0 01 second VA vector may assume in

the R wave in leads V_6 and I Because of the delayed onset of left ventricular excitation and the delayed appearance of the maximal instantaneous vector onset of the intrinsicoid deflection in lead V_6 is likewise delayed to 0 08–0 10 second after the beginning of the QRS interval

Leads aVF and V_1.—The 0 08 second VA vector coincides with the nadir of the deep wide S wave in these leads

Precordial QRS transition.—Since the maximal instantaneous vector is oriented farther posteriorly than normal (and the maximal mean instantaneous QRS spatial vector corresponds roughly to the calculated mean QRS spatial vector) the transitional chest lead whose axis is perpendicular to the maximal vector is located farther to the left than in the normal precordial electrocardiogram Thus in left bundle branch block leads V_1 through V_4 or V_5 usually register resultantly negative or downwardly directed QRS deflections while lead V_6 records a broad R wave Although the right precordial leads may show QS deflections in left bundle branch block such deflections probably do not occur as often as previously thought Grant and Dodge found that 45% of their patients with left bundle branch block had electrocardiograms showing initial R waves in leads V_1 through V_4 The R waves were absent in lead V_1 in only 35% of the electrocardiograms in 15% the R waves were absent in leads V_1 and V_2 in only 5% were R waves absent in leads V_1 through V_3 and in none of the electrocardiograms were R waves absent as far to the left as V_4 This was in agreement with our observations in patients with left bundle branch block 75% of the electrocardiograms showed initial R waves in all precordial leads in the remaining 25% with QS deflections in lead V_1 initial R waves were present in lead V in about half of the cases In our experience QS deflections in lead aVF were noted relatively uncommonly

0 10-Second VA Vector

This vector probably is related to activation of the anterolateral wall of the left ventricle and is therefore of smaller magnitude than the 0 08-second vector and is directed less posteriorly and superiorly

Leads I and V_6.—Downstroke of the broad R wave

Leads aVF and V_1.—Terminal limb of the S wave

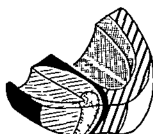
The late appearance of the 0 10 second and subsequent vectors because of delayed onset of left ventricular depolarization obviously leads to prolongation of the QRS interval and so the QRS duration in left bundle branch block is 0 12 second or longer

VENTRICULAR REPOLARIZATION

Shortly after the excitation impulse enters and begins to spread through the left ventricular myocardium recovery commences in regions of the ventricles first activated notably in the interventricular septum (Repolarization potentials generated by the free wall of the right ventricle are negligible and may for all intents and purposes be ignored) Recovery starts first at the right septal surface and then the repolarization wave spreads from right to left through the muscle mass of the septum The septal repolarization forces thereby produced are directed to the right and anteriorly just the reverse of the septal depolarization

Fig 141 —
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A Sequence of Septal Ventricular Activation in Left Bundle Branch Block

1 INITIAL ACTIVATION OF APICO ANTERIOR RIGHT VENTRICULAR WALL

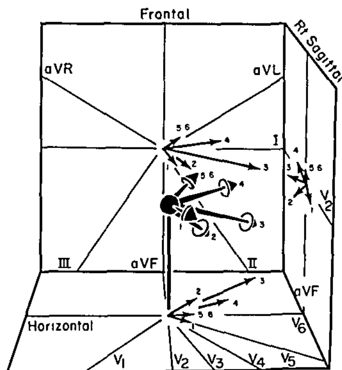
2 RIGHT-TO LEFT SEPTAL ACTIVATION AND ACTIVATION OF RIGHT VENTRICULAR FREE WALL

3 COMPLETION OF SEPTAL AND RIGHT VENTRICULAR ACTIVATION

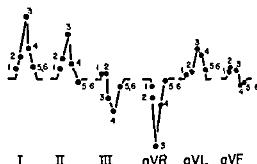
4 INITIAL ABERRANT ACTIVATION OF BASAL LEFT VENTRICULAR WALL

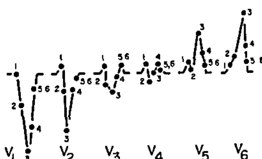
5 ACTIVATION OF POSTERIOR LATERAL AND ANTERIOR LEFT VENTRICULAR WALL

6 COMPLETION OF ACTIVATION OF ANTERIOR WALL OF LEFT VENTRICLE

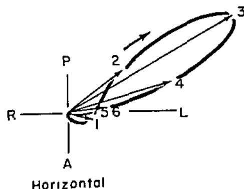


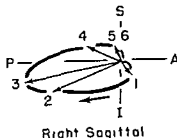
B Instantaneous VA Vectors in Left Bundle Branch Block

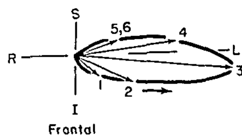




C QRS Deflections Projected on Scalar Leads







D Planar QRS Loops in Left Bundle Branch Block

Fig 141 —(*Legend on facing page*)

VECTORCARDIOGRAPHIC FINDINGS

QRS sÊ Loop Abnormalities

The following abnormal changes were noted in the vectorcardiogram (see also Fig 142 and Table 17

Horizontal QRS loop — In about one third of our cases of left bundle branch block the horizontal QRS loop was written initially to the left and anteriorly in an equal number of cases the loop proceeded initially to the left and posteriorly while in slightly less than a third of the cases the horizontal loop was inscribed anteriorly and slightly to the right The rest of the horizontal QRS loop whatever the direction of the initial deflection was inscribed in a clockwise direction [illegible]

horizontal reference frame In the afferent limb of the loop or occasionally in its midportion the time markings were closely spaced indicating conduction delay After its inscription the horizontal QRS loop did not return to its point of origin instead its junction with the T loop was displaced to the right and anteriorly indicating a similarly directed ST vector

Occasionally in left bundle branch block the horizontal QRS loop has a figure-of-eight configuration the distal loop of the "eight" usually being written in a clockwise direction In loops of this contour the initial and early vectors generally lie anteriorly and to the left

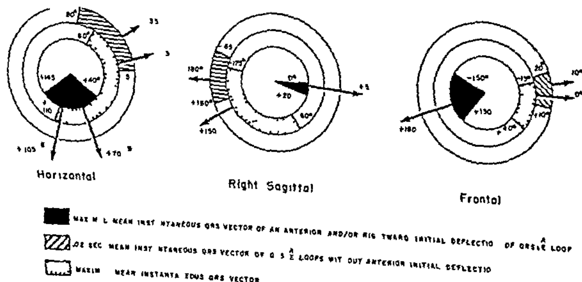
Right sagittal QRS loop — The sagittal QRS loop is almost invariably written in a clockwise direction posteriorly and either slightly inferiorly or superiorly its long axis ordinarily lying between +170 and −175 in the sagittal reference frame The afferent limb the midportion of the loop or both are inscribed slowly and the junction of the afferent limb with the T loop is displaced anteriorly and slightly inferiorly

Frontal QRS loop — The frontal QRS loop is usually written in a counterclockwise direction and situated within the −15 to 0 segment of the frontal reference frame An S–T vector directed to the right and inferiorly is often present

T sÊ Loop

The spatial T loop is discordant to the QRS sÊ loop Its general orientation is to the right anteriorly and either superiorly or inferiorly (Figs 143–145)

Fig 142 — Extreme range of variation and av[illegible]



forces in left bundle branch block Since the instantaneous T vectors make their appearance before the completion of left ventricular activation the terminal *limb of the QRS deflection does not stop at the base* line but continues to rise or descend as the case may be until it merges with an elevated or depressed S–T segment The S–T segment in a given lead is displaced in the direction of the abnormal T wave since both abnormalities are manifestations of the same process namely altered ventricular repolarization In effect it is as if an S–T vector exists which is oriented parallel to the instantaneous T vectors

Since the abnormal angular divergence of the instantaneous VA (or QRS) and T vectors in left bundle branch block is due fundamentally to the abnormal spread of excitation through the ventricle the T waves and S–T segments of the electrocardiogram in left bundle branch block display *secondary changes* but the ventricular gradient remains normal Other mechanisms (related to the gradient concept) which may contribute to the genesis of the secondary T wave changes in left bundle branch block are as follows

1 According to the ventricular gradient concept $\hat{A}$ QRS + $\hat{A}$ T ≈ G or $\hat{A}$ T = G − $\hat{A}$ QRS If the mean ventricular gradient vector G remains normal then $\hat{A}$ T ≈ − $\hat{A}$ QRS

In other words every change in the area of the QRS complex causes a corresponding secondary change in the area of the ST–T complex which is equal in magnitude but opposite in direction to the QRS change Therefore the greater the positive area *of the QRS complex in left precordial leads the more* marked will be the S–T segment depression and T wave inversion Similarly the greater the negative area of the QRS complexes in right precordial leads *the more prominent the S–T segment elevation and* the taller the T waves

2 *If* as some authorities believe in left bundle *branch block there* is aberrant spread of excitation through the left ventricular wall then the direction of repolarization may be reversed in the left ventricle as well as in the septum

ELECTROCARDIOGRAPHIC FINDINGS

The electrocardiographic findings in left bundle branch block are listed below

EXTREMITY LEADS

1 QRS interval prolongation to 0 12 second or longer

2 *Leads I and aVL Absent Q waves and slurred* broad R waves

3 Leads II III and aVF rS deflections with deep wide S waves (occasionally QS deflections)

4 Left axis deviation of $\hat{A}$ QRS the orientation of the latter ranging from −60 to 0 in the frontal reference frame

PRECORDIAL LEADS

1 Delayed onset of the intrinsicoid deflection in lead V_6 (to 0 05 second or sometimes 0 08–0 10 second) normal intrinsicoid deflection time in lead V_1

2 Lead V_6 *Absent* Q wave and slurred broad R wave

3 Leads V_1 and V_2 rS deflections with deep wide S waves (occasionally QS deflections)

Leads V_3 and V_4 rS or RS (rarely QS) deflections Usually there is a progressively increasing R/S amplitude ratio from right to left across the left precordium in left bundle branch block but sometimes the R waves in the midprecordial leads may be relatively smaller than those in the right precordial leads

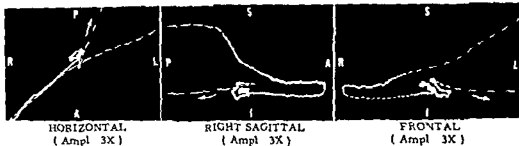
4 Posterior rotation of $\hat{A}$ QRS the orientation of the latter tending to range from −60 to −30 in the horizontal reference frame

5 S–T vector and s$\hat{A}$ T are usually almost 180 discordant to s$\hat{A}$ QRS Therefore

a) Leads registering upright R waves (I aVL and V_6) generally display depressed S–T segments and inverted T waves

– – downwardly directed ventricular deflections (II III aVF and V through V es with a tendency to superior displacement of junction J of the S–T *segment*

iation and the size of the T waves vary directly with the area enclosed by the



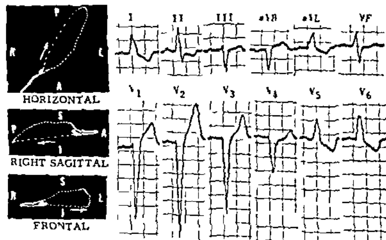


Fig 143 —Electro...

TABLE 17 — VECTORCARDIOGRAPHIC FINDINGS IN LEFT BUNDLE BRANCH BLOCK

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
Orientation of the maximal mean instantaneous QRS vector	−60 to +110	−15	−60 to −30	+60 to −175	+150	+170 to −175*	−15 to +40	0	
Orientation of mean 0 02 second instantaneous QRS vector	−80 to −5	−35	−50 to −20	+160* to −165	180	+170 to −175	−20 to +10*	−10	−20 to 0
Orientation of maximal vector of anterior and/or rightward initial deflection of the QRS sÊ loop (when present)	+40 to +145	+105 +70 †	+80 to +100	0 to +20	+5		+130 to −150	+160	
Direction of inscription of									
a) Initial deflection of QRS sÊ loop (if present)	1 Counterclockwise if initial deflection is directed to the left 2 Clockwise if initial deflection is directed to the right			Usually clockwise			Usually counterclockwise		
b) Efferent and afferent limbs of QRS sÊ loop	Clockwise			Clockwise			Counterclockwise		
Direction of S-T vector	Right anterior			Anterior inferior			Right inferior		
Direction of initial inscription of QRS sÊ loop in horizontal projection	Anterior leftward 35%‡ Anterior rightward 30%‡ Posterior leftward 35%‡								
Time of onset of maximal vector of horizontal QRS loop	Extreme Range: 0 015–0 10 second	Av: 0 065 second	Usual Range: 0 05–0 09 second						
Orientation of T sÊ loop	Usually approximately 180 discordant to the long axis or maximal mean vector of the QRS sÊ loop								

* Average orientation of rightward and anterior initial deflection of QRS sÊ loops in left bundle branch block
† Average orientation of leftward and anterior initial deflection of QRS sÊ loop in left bundle branch block
‡ The percentage of vectorcardiograms showing left bundle branch block in which the initial deflection of the QRS sÊ loop was directed

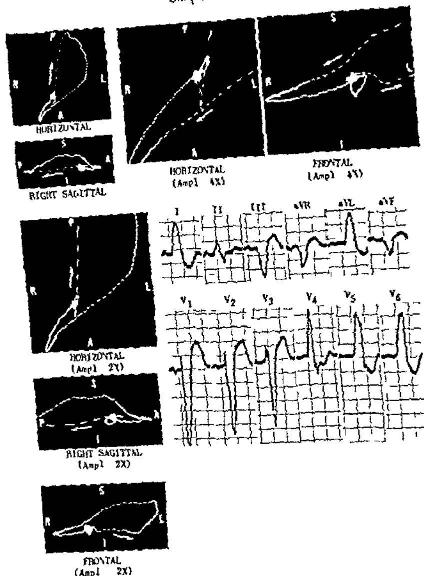


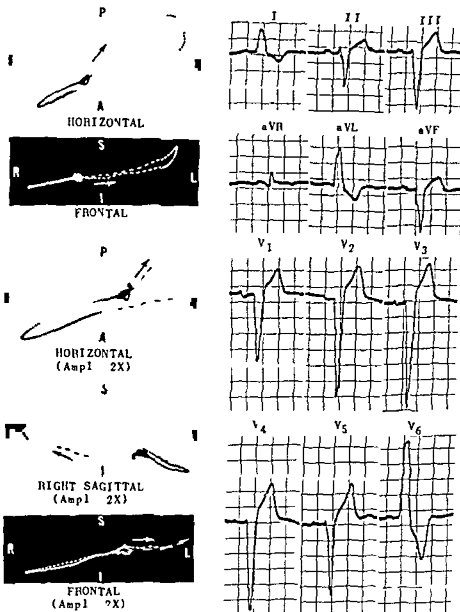
Fig 145 – ...
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INCOMPLETE LEFT BUNDLE BRANCH BLOCK

Electrocardiographic Findings

It will be recalled that the principal point of distinction (arbitr ry though it may be) which has been made in the past between complete and incomplete bundle branch block is that the QRS duration is 0 12 second or more in complete block and less than 0 12 second in incomplete block Although there is some variation in the electrocardiographic criteria listed by different investigators for the diagnosis of incomplete left bundle branch block on the whole Barker s criteria can be considered representative The features which Barker regards as suggestive of incomplete left bundle branch block are as follows

1 The QRS interval is less than 0 12 second and at least 0 10 second in duration
2 The QRS complexes do not usually show high voltage
3 There are no Q waves in leads V_5 and V_6



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sis of left ventricular hypertrophy cannot be made in the presence of left bundle b ... the increased QRS voltages are not necessarily related to the thickness of the left ventricular wall but may reflect aberrant spread of activation through the left ventricle

4 There is slurring or notching of the QRS complexes such as is seen in complete left bundle branch block

Sodi Pallares and his associates have extensively studied incomplete left bundle branch block produced experimentally in the dog heart and from these in

which has already been discussed The electrocardiographic characteristics of first and second-degree (incomplete) left bundle branch block as described by Sodi Pallares and his co-workers are as follows

First-degree (incomplete) left bundle branch block —This degree of block has the following characteristics (see also Fig 146)

1 Initial slurring of the R wave in leads V_5 V_6 and/or lead I generally associated with absence of Q waves or with diminutive Q waves in these leads
2 QRS duration ranging from 0 07 to 0 12 second, with the greater majority of cases having QRS in

3

st cases

Second-degree (incomplete) left bundle branch block —Characterized as follows

1

2 QRS duration from 0 12 to 0 15 second

In first-degree (incomplete) left bundle branch block Sodi Pallares and his associates postulate that the functional capacity of the left bundle branch is only slightly affected and that the excitation impulse is conducted down the left branch but at a slower rate than normally The normal time lag of right septal activation after onset of excitation on the left septal surface tends therefore to be abolished in incomplete left bundle branch block with the result

ing muscle the apical portion of the

that the initial forces due to left to right septal depolarization are opposed and in part at least neutralized This circumstance is responsible for the absence or decreased size of the Q wave in leads I and V_6 and the slurring of the early upstroke of the R wave in these leads In second-degree (incomplete) left bundle branch block Sodi Pallares interprets the experimental findings as indicating that the left bundle branch is still functioning but at a very slow rate of conduction Consequently while the left branch activates some of the muscle mass of the left septum the remainder receives the excitation impulse transseptally from the right bundle branch

Vectorcardiographic Findings

QRS sÊ Loop Abnormalities

The vectorcardiographic findings in incomplete left bundle branch block have not as yet been described in any great detail According to Sodi Pallares in first-degree (incomplete) left bundle branch block the QRS sÊ loop in the horizontal projection is inscribed in the normal counterclockwise direction but with some delay in early portions of the loop while in second degree (incomplete) left bundle branch block the QRS sÊ loop in the horizontal projection can be inscribed in either a counterclockwise or a clockwise direction From the brief descriptions of other investigators it would seem that the vectorcardiographic findings in incomplete left bundle branch block resemble those in complete left bundle branch block except for the duration of the QRS sÊ loop

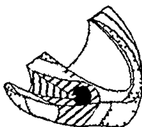
The authors of this text studied the vectorcardiograms of a relatively small group of patients whose electrocardiograms provided the basis for selection All of the following requirements were satisfied by the electrocardiograms of the cases selected

1 The R waves in leads I and V sho ... or

2

3 ... cases showed no definite evidence of anterior infarction

The frontal



■ 1 INITIAL EXCITATION NEAR BASE OF RIGHT ANTERIOR PAPILLARY MUSCLE VIA RIGHT BUNDLE BRANCH (RBB)

▨ 2 EXCITATION OF APICO ANTERIOR VENTRICULAR FREE WALL AND PREDOMINANT RIGHT-TO LEFT SEPTAL EXCITATION VIA RIGHT BUNDLE BRANCH

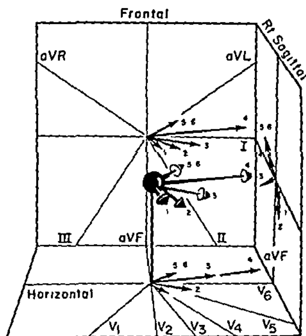
□ 3 EXCITATION OF SEPTUM IN RIGHT TO LEFT DIRECTION AND APICO ANTERIOR WALL ACTIVATION VIA BOTH BUNDLE BRANCHES

□ 4 ACTIVATION OF ANTEROLATERAL LEFT VENTRICULAR WALL AND RIGHT VENTRICULAR WALL

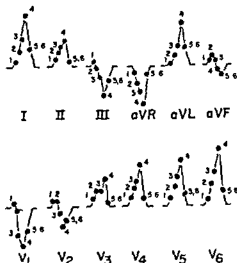
□ 5 ACTIVATION OF POSTEROLATERAL WALL OF LEFT VENTRICLE

□ 6 ACTIVATION OF POSTEROBASAL LEFT VENTRICULAR WALL

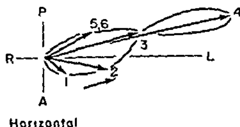
A Sequence of Septal Ventricular Activation in Incomplete Left Bundle Branch Block

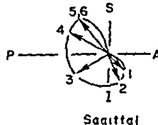


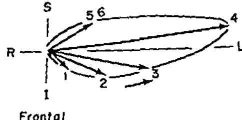
B Instantaneous VA Vectors in Incomplete Left Bundle Branch Block



C QRS Deflections Projected On the Scalar Leads







D Planar QRS Loops in Incomplete Left Bundle Branch Block

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4 There is slurring or notching of the QRS complexes such as is seen in complete left bundle branch block.

Sodi Pallares and his associates have extensively studied incomplete left bundle branch block produced experimentally in the dog heart and from these investigations they have formulated a classification of left bundle branch block into three degrees the third degree being complete left bundle branch block which has already been discussed The electrocardiographic characteristics of first and second-degree (incomplete) left bundle branch block as described by Sodi Pallares and his co-workers are as follows

First-degree (incomplete) left bundle branch block —This degree of block has the following characteristics (see also Fig 148)

1 Initial slurring of the R wave in leads V₅ V₆ and/or lead I generally associated with absence of Q waves or with diminutive Q waves in these leads
2 QRS duration ranging from 0 07 to 0 12 second with the greater majority of cases having QRS intervals between 0 08 and 0 11 second
3 Onset of intrinsicoid deflection in lead V₆ later than 0 045 second after start of QRS interval in most cases

Second degree (incomplete) left bundle branch block —Characterized as follows

1 Slurring involves not only the initial portion of the R wave but also the ascending limb of the R wave and occasionally the descending limb
2 QRS duration from 0 12 to 0 15 second

In first-degree (incomplete) left bundle branch block Sodi Pallares and his associates postulate that the functional capacity of the left bundle branch is onl[illegible]

[illegible] activation after onset of excitation on the left septal surface tends therefore to be abolished in incomplete left bundle branch block with the result that the initial forces due to left to right septal depolarization are opposed and in part at least neutralized This circumstance is responsible for the absence or decreased size of the Q wave in leads I and V₆ and the slurring of the early upstroke of the R wave in these leads In second degree (incomplete) left bundle branch block Sodi Pallares interprets the experimental findings as indicating that the left bundle branch is still functioning but at a very slow rate of conduction Consequently while the left branch activates some of the muscle mass of the left septum the remainder receives the excitation impulse transseptally from the right bundle branch

Vectorcardiographic Findings

QRS sÊ Loop Abnormalities

[illegible]

the QRS sÊ loop in the horizontal projection is inscribed in the normal counterclockwise direction but with some delay in early portions of the loop while in second-degree (incomplete) left bundle branch block the QRS sÊ loop in the horizontal projection can be inscribed in either a counterclockwise or a clockwise direction From the brief descriptions of other investigators it would seem that the vectorcardiographic findings in incomplete left bundle branch block resemble those in complete left bundle branch block except for the duration of the QRS sÊ loop

The authors of this text studied the vectorcardiograms of a relatively small group of patients whose electrocardiograms provided the basis for selection All of the following requirements were satisfied by the electrocardiograms of the cases selected

1 The R waves in leads I and V₆ showed slurred or notched upstrokes while Q waves were absent
2 The QRS duration was less than 0 12 second
3 The precordial leads showed no definite evidence of anterior infarction

ing muscle the apical portion of the interventricular septum and the anterior free wall of the right ventricle are undergoing activation The resultant of the electrical forces arising in th[illegible] tricle corresponds to vector [illegible] left bundle branch [illegible] wall of the left v[illegible]

The requirements listed above contain some of the criteria both of Barker and of Sodi Pallares for the diagnosis of incomplete left bundle branch block The additional requirements were intended to exclude in so far as possible anteroseptal infarction which may mimic incomplete left bundle branch block The indication of left ventricular hypertrophy in the electrocardiogram was not considered a basis for excluding a case from the study series since this condition seemed to be associated so frequently in the electrocardiogram with the features of incomplete left bundle branch block that there seemed to be some relationship between these two entities

There was found to be surprising consistency in *the vectorcardiographic findings in our small group* of patients the salient abnormalities being as follows

HORIZONTAL QRS LOOP —The long axis of the QRS sÊ loop in the horizontal projection usually is situated between the −45° and −15 axes of the horizontal reference frame The loop is written initially to the left and anteriorly In about half of the vectorcardiograms showing incomplete left bundle branch block the initial and early segments of the loop are inscribed somewhat slowly The loop then is written to the left and posteriorly in the form of a figure of eight the proximal loop being counterclockwise inscribed Occasionally there is conduction delay in the very terminal portion of the loop

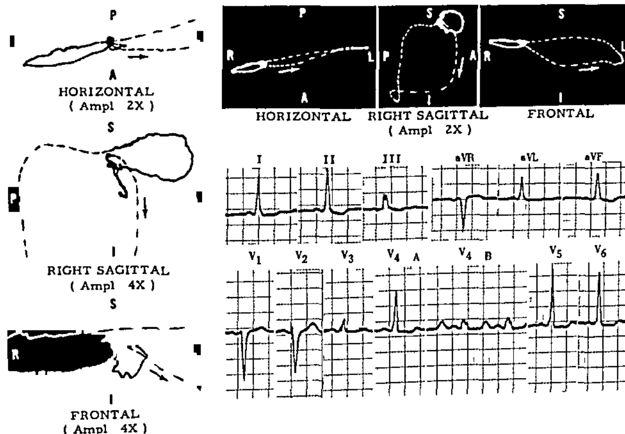
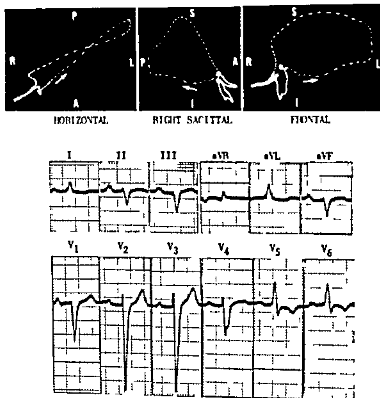


Fig 147 —Electrocardiographic and vectorcardiographic patterns of incomplete left bundle branch block
The l ... abnormalities in the elec

QRS sÊ loop

Fig 148 — Electrocardiographic and vectorcardiographic patterns of incomplete left bundle branch block The QRS duration in the electrocardiogram is 0 12 second but the configuration of the QRS deflections is more in keeping with incomplete than with complete left bundle branch block The slurring of the R waves in leads I and aVL occurs on their upstrokes rather than at their summits and terminal S waves are present in leads V and V The QRS sE loop of the vectorcardiogram shows a left posterior initial deflection with slowed inscription of this and the adjacent segment of the efferent limb of the loop The horizontal QRS loop has a figure-of-eight configuration and the frontal loop is situated superiorly and to the left



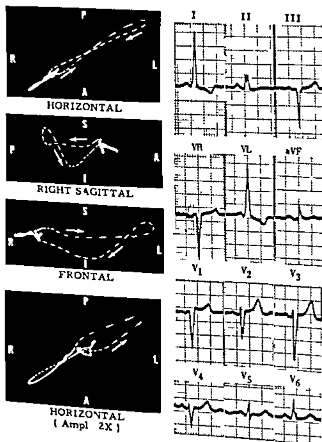


Fig 149 — Electrocardiographic and vectorcardiographic patterns of incomplete left bundle branch block In the vectorcardiogram the counterclockwise inscription of the sagittal QRS loop and the clockwise inscription of the frontal loop are uncommon findings in incomplete left bundle branch block the significance of which is not known

RIGHT SAGITTAL QRS LOOP —The sagittal loop is written posteriorly and slightly inferiorly or superiorly The initial deflection of the loop is usually directed slightly anteriorly but sometimes posteriorly In a small percentage of the vectorcardiograms the sagittal loop is counterclockwise inscribed although clockwise inscription is by far the more common finding *Conduction delay may be present in the early* portion of the loop in some instances and in the afferent limb of the loop in others

FRONTAL QRS LOOP —The frontal QRS loop tends to lie almost horizontal and to the left The direction of inscription varies some loops being clockwise and others counterclockwise inscribed By and large counterclockwise inscription of the frontal loop is the usual finding

S–T VECTOR

An S–T vector is observed frequently and is usually directed to the right anteriorly and superiorly or inferiorly

T sÊ LOOP

The T sÊ loop is almost always discordant to the QRS *sÊ loop and more or less parallel to the S–T* vector (Figs 147–149)

It should be stressed that the vectorcardiographic features ascribed above to left bundle branch block represent preliminary tentative findings the validity of which awaits further study

VARIANT TYPE OF LEFT INTRAVENTRICULAR BLOCK

Vectorcardiographic Findings

A variant pattern frequently encountered which exhibits a high degree of consistency in its features is the type described by Grishman and his associates This pattern they feel is indicative of left ventricular hypertrophy with terminal conduction delay As observed by us this pattern was characterized in the vectorcardiogram by the following features

HORIZONTAL PROJECTION

QRS sÊ LOOP —The horizontal QRS loop varied in orientation within the range of −75 to −30 (average orientation −60°) There usually was an initial deflection of the loop to the right and anteriorly (occasionally to the left and anteriorly) and then the remainder of the loop was written in a counterclockwise direction to the left and farther posteriorly than normal The returning or afferent limb of the loop invariably showed conduction delay and was often situated *somewhat to the right Frequently the QRS sÊ* loop in the horizontal projection remained open the S–T vector being directed to the right and anteriorly

T sÊ LOOP —*The long axis of the horizontal T loop* was almost invariably discordant to the long axis of the QRS loop The average orientation of the T sÊ *loop in the horizontal projection was along the +95* axis of the horizontal reference frame

RIGHT SAGITTAL PROJECTION

QRS sÊ LOOP —The sagittal loop was usually written *in a clockwise direction* anteriorly and inferiorly and then posteriorly and superiorly Again the afferent *limb showed closely spaced dashes indicating conduction delay* The orientation of the long axis of the sagittal QRS loop ranged between −170 and −120° (average orientation −140)

T sÊ LOOP —The T loop was discordant to the QRS loop in this projection and was situated on the average along the +30 axis of the sagittal reference frame

FRONTAL PROJECTION

QRS sÊ LOOP —The major portion of the frontal QRS loop usually was located in the left upper quadrant the long axis of the loop ranging between −75° and 0° (average orientation −40) The loop was *usually written in a counterclockwise direction* with the afferent limb showing conduction delay and frequently lying to the right of the midline

T sÊ LOOP —The T loop was discordant to the QRS loop its average orientation being along the +120 axis of the frontal reference frame

Electrocardiographic Findings

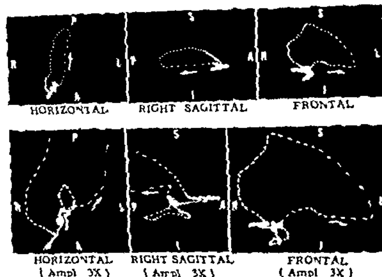
In cases with the vectorcardiographic variant pattern just described the electrocardiogram typically showed the following findings

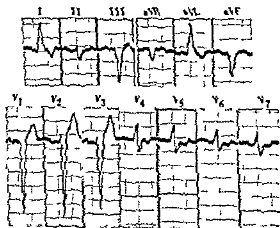
1 There is marked left axis deviation of Â QRS in the bipolar limb leads the manifest electrical axis of QRS lying on the average along the −55° axis of the frontal reference frame
2 The QRS duration varies between 0 11 and 0 15 second

Fig 150 — Electrocardiographic and vectorcardiographic findings in the variant type of left intraventricular block designated by Grishman and his associates as "left ventricular hypertrophy with terminal conduction delay."

The most characteristic feature of the electrocardiogram in this form of intraventricular block is the wide slurred terminal S wave in lead V. Additional findings usually observed are left axis deviation of $\hat{A}$ QRS, prolonged QRS interval and secondary S–T segment and T wave changes.

The vectorcardiographic abnormalities are marked posterior leftward and superior rotation of the QRS $s\hat{E}$ loop, conduction delay involving all or only the terminal portion of the afferent limb of the QRS $s\hat{E}$ loop and inscription of this part of the QRS $s\hat{E}$ loop to the right posteriorly and superiorly. The latter inscription is responsible for the terminal S wave in lead V. The direction of inscription of the QRS $s\hat{E}$ loop is normal in each projection. The initial deflection of the QRS $s\hat{E}$ loop in the above vectorcardiogram is to the left, but not infrequently in this form of left intraventricular block the initial deflection of the QRS $s\hat{E}$ loop can be written to the right and anteriorly.







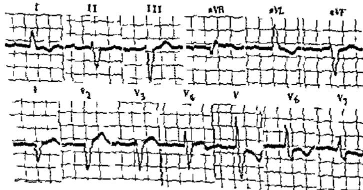
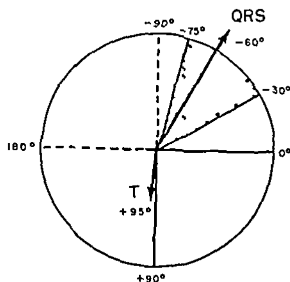
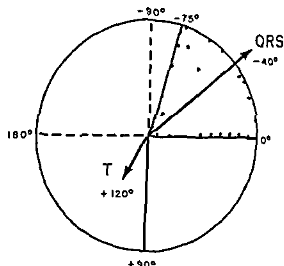


Fig 151 — Electrocardiographic and vectorcardiographic patterns of the variant type of left intraventricular block ("left ventricular hypertrophy with terminal conduction delay")





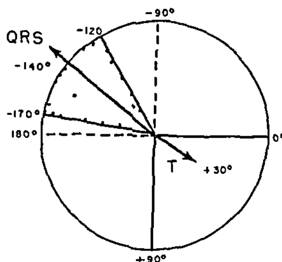


Fig 152 —Range of variation and average orientation of the maximal mean instantaneous QRS vector and maximal mean instantaneous T vector of the vectorcardiogram in the variant type of left intraventricular block (*left ventricular hypertrophy with terminal conduction delay*)

3 Because of the marked posterior rotation of the QRS sÊ loop the precordial lead recording the transitional RS deflection is shifted far to the left producing the electrocardiographic pattern of marked clockwise rotation

Thus rS deflections are present in most leads to the right of lead V_6 which usually shows an RS deflection with the S wave being relatively wide Occasionally rS deflections are recorded in leads as far to the left as V_8 despite the fact that lead I displays a relatively taller R wave Such cases as these in which leads I and V_6 register QRS deflections of differing configuration (qR or R in lead I and RS in lead V_6) may be explained as follows

Lead I —Typically in vectorcardiograms showing this type of intraventricular conduction disturbance the frontal QRS loop is located almost entirely superiorly and the terminal limb of the loop may even extend slightly to the right of the electrical null point Despite this fact lead I records terminal positivity because its effective lead axis is slanted downward from left to right Thus as long as a vector is located *superiorly* it may even lie slightly to the right and still project on the positive half of the axis of derivation of lead I

Lead V_6 —In contrast with lead I lead V_6 has an effective axis which slants upward from left to right and this being the case superiorly oriented vectors

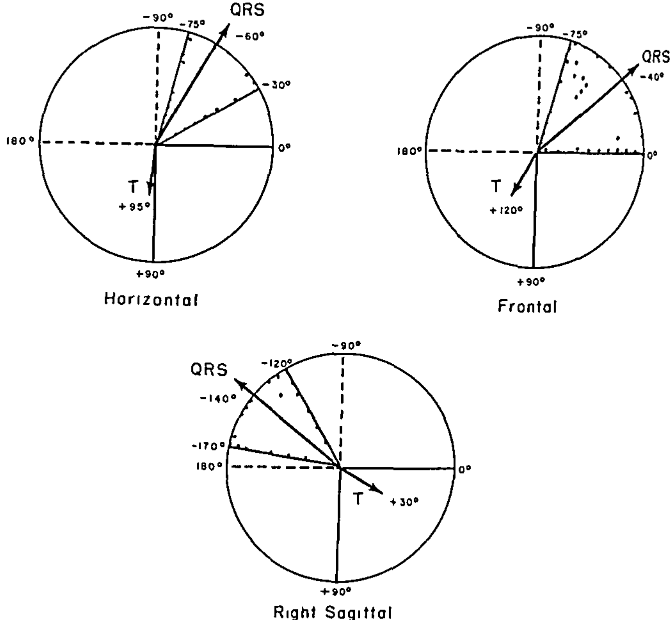


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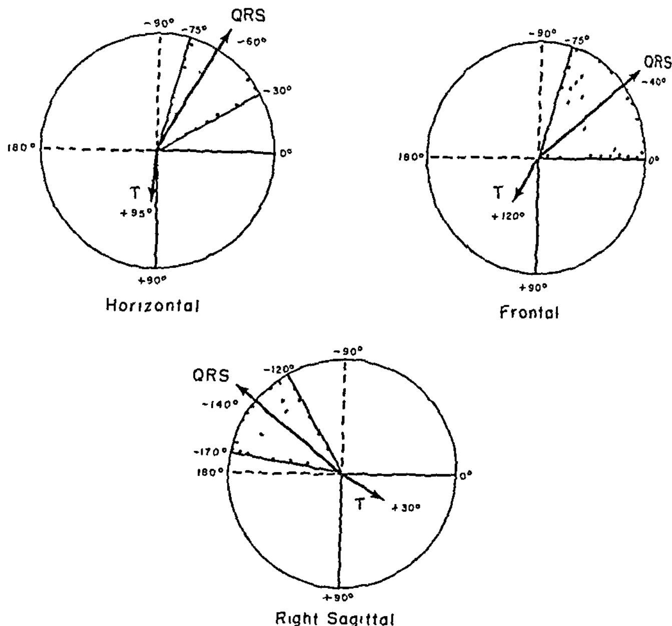


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Block located peripheral to the main right bundle branch —The effects of a peripheral or focal block of right intraventricular conduction on the ventricular activation process have been explained in two ways

1 Segers and his associates and Vastesaeger propose that a localized region of the right ventricle receives the excitation impulse via the myocardial fibers rather than the Purkinje system Activation of this region begins late in the QRS interval and proceeds slowly

2 Dodge and Grant believe that in most (but not all) cases of right bundle branch block the site of the block is peripheral and that not all of the right ventricle is affected by the block These investigators postulate that as soon as excitation reaches the site of the block it immediately spreads out into the surrounding regions of the right ventricle as if the lesion were a leak for excitation rather than a delaying or blocking mechanism The remainder of the right ventricle is depolarized by abnormal routes and slower than normal velocities

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In fact there is reason to believe that the pattern of classic right bundle branch block may be related etiologically to myocardial infarction involving several aspects of the left ventricle and occurring in company with an intraventricular conduction disturbance other than right bundle branch block Thus even aside from the rarity of the classic right bundle branch block pattern the uncertainty regarding its mechanism and significance would seem to be reason enough for discussing it separately apart from the bona fide right bundle branch block patterns to be described shortly In its place we have substituted what for want of better terminology will be referred to hereafter as the *right bundle branch block variant pattern* While fundamentally the common and variant types of right bundle branch block exhibit essentially similar identifying features in the electrocardiogram and vectorcardiogram nevertheless the two patterns differ sufficiently from each other in their electrical effects as well as in their significance and the clinical circumstances in which they appear to justify making a distinction between them Moreover as will be explained later the level of the block in the right intraventricular conducting pathways in the right bundle branch block variant may not be the same as that in the common type of right bundle branch block

The common type of right bundle branch block can be considered the electrocardiographic and vectorcardiographic prototype to which most of the pattern variations observed in clinical electrocardiography tend in general to conform In the following pages however the instantaneous VA vectors and the electrocardiographic and vectorcardiographic findings in both the common and the variant types of right bundle branch block will be described in parallel To avoid the confusion that might ensue from such a complicated presentation the salient characteristics *common* to both types of right bundle branch block are herewith listed briefly

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Thus The first 0 06 second of the electrocardiographic QRS deflection and the vectorcardiographic QRS sÊ loop are relatively normal in appearance or show the same abnormalities as they

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2 During the second half of ventricular activation (i e during the terminal 0 04–0 06 second of the QRS interval) abnormal QRS forces directed to the right and anteriorly are produced for a period after the time when ventricular activation would normally have been completed

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THE INSTANTANEOUS VA VECTORS IN COMMON AND VARIANT TYPES OF RIGHT BUNDLE BRANCH BLOCK

The spread of activation through the interventricular septum and ventricles in right bundle branch block and the electrical forces produced can be presented in simplified manner in terms of the instantaneous VA vectors (see also Fig 154)

0 01 Second Septal VA Vector

Since the left bundle branch is intact *the left* septal surface and the greater thickness of the septal muscle mass are usually activated *just as during nor*mal intraventricular conduction However in occasional cases of right bundle branch block, the initial mean instantaneous spatial vectors of the vectorcardiographic QRS sE *loop* are found to be directed to the left and slightly anteriorly or *less commonly* slightly posteriorly While the abnormal orientation *of the initial* vectors *in* such cases can sometimes be attributed to antecedent myocardial infarction this *leaves unexplained* the instances in which there is neither electrocardiographic nor clinical evidence of old or *recent infarction* At present, information is too incomplete to permit anything other than speculation as to the mechanism responsible for the leftward and slightly anterior or posterior initial vectors in some cases of right bundle branch block One possible

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In fact there is reason to believe that the pattern of classic right bundle branch block may be related etiologically to myocardial infarction involving several aspects of the left ventricle and occurring in company with an intraventricular conduction disturbance *other than right bundle branch block Thus* even aside from the rarity of the classic right bundle branch block pattern the uncertainty regarding its mechanism and significance would seem to be reason enough for discussing it separately apart from the bona fide right bundle branch block patterns to be described shortly In its place, we have substituted

ant types of right bundle branch block exhibit essentially similar identifying features in the electrocardiogram and vectorcardiogram nevertheless the two patterns differ sufficiently from each other in their electrical effects as well as in their significance and the clinical circumstances in which they appear to justify making a distinction between them Moreover as will be explained later *the level of the block* in the right intraventricular conducting pathways in the right bundle branch block variant may not be the same as that in the common type of right bundle branch block

The common type of right bundle branch block can be considered the electrocardiographic and vectorcardiographic prototype to which most of the pattern variations observed in clinical electrocardiography tend in general to conform In the following pages however the instantaneous VA vectors and the electrocardiographic and vectorcardiographic findings in both the common and the variant types of right bundle branch block will be described in parallel To avoid the confusion that might ensue from such a complicated presentation the salient characteristics *common* to both types of right bundle branch block are herewith listed briefly

1 Approximately the first half of ventricular activation is usually not altered significantly by the presence of right bundle branch block

Thus The first 0 06 second of the electrocardiographic QRS deflection and the vectorcardiographic *QRS sÊ loop are relatively* normal in appearance or show the same abnormalities as they

0.02 Second Apicoanterior VA Vector

Because the excitation impulse is blocked (or delayed) in its passage down the right bundle branch the septum continues to be activated from left to right. However at the same time excitation is transmitted in a normal fashion to the left ventricle where ... 02 ... nd after onset of ventricular de-

and direction as those produced during normal intraventricular conduction, at least in the common type of right bundle branch block. For one reason or another—whether due to concomitant ventricular hypertrophy and/or myocardial damage or to the site of the block in the right bundle branch system—the 0.02 second VA vector in the variant type of right bundle branch block is directed superiorly to the left and slightly anteriorly and may be of smaller than normal magnitude.

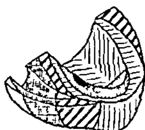
	Common Type of Right Bundle Branch Block	Variant Type of Right Bundle Branch Block
	0.02 second VA vector directed to the left anteriorly and inferiorly	0.02 second VA vector directed to the left anteriorly and superiorly
Leads I and V_6	Beginning upstroke of an R wave.	Upstroke of an R wave
Lead aVF	Upstroke of an R wave	Beginning downstroke of an S wave
Lead V_1	Completion of small initial R wave	Completion of small initial R wave or the beginning upstroke of a slurred R wave or of an R wave following a small Q wave

0.04-Second Left Ventricular VA Vector

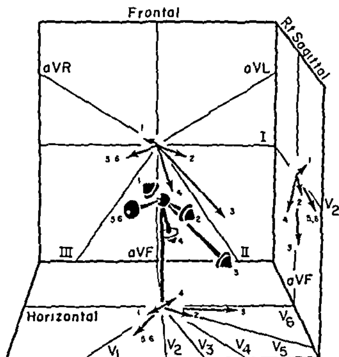
Activation of the free wall of the left ventricle gives rise to forces which dominate those produced by continuing left-to-right septal depolarization. In fact, the electrical predominance of the left ventricle is maintained for approximately the first 0.04 second of the QRS interval only, the 0.04 second VA vector being the maximal leftward mean instantaneous vector produced during ventricular activation in right bundle branch block just as normally. The orientation and magnitude of the 0.04 second VA vector may differ little from the normal, particularly in the common type of right bundle branch block, but often in the variant type of right bundle branch block, for reasons unknown, the 0.04 second vector is of lesser magnitude and more superior orientation than the corresponding normal vector or that occurring in the common type of right bundle branch block.

	Common Type of Right Bundle Branch Block	Variant Type of Right Bundle Branch Block
	0.04 second VA vector directed to the left, posteriorly and inferiorly	0.04 second VA vector directed to the left superiorly and usually posteriorly, occasionally anteriorly
Leads I and V_6	Completion of upstroke of R wave. The 0.04 second VA vector roughly coincides with ...	Because of the marked superior orientation of the 0.04 second VA vector in this type of right bundle branch block pattern the projection of the vector on the transverse lead axes of leads I and V_6 is much less than that of the corresponding vector in the common type of right bundle branch block. Thus the peak of the R in leads I and V_6 is relatively low

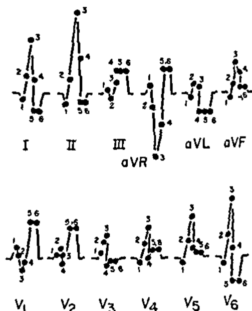
Fig. 154.—Instantaneous VA vectors in right bundle branch block. In B instantaneous VA vector 5.6 is so numbered to indicate that it actually represents several successive instantaneous vectors having much the same direction and magnitude. These combined vectors correspond to the late slowly inscribed R′ wave of the RSR′ deflection in lead V_1 of C.



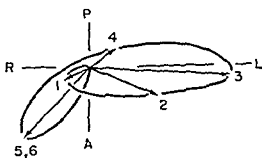
A Sequence of Septal-Ventricular Activation in Right Bundle Branch Block

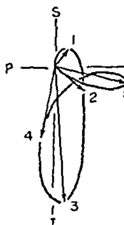


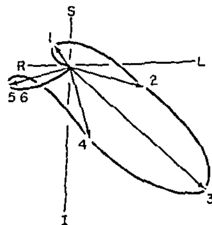
B Instantaneous VA Vectors in RBBB



C QRS Deflections Projected on Scalar Leads







D Planar QRS Loops in Right Bundle Branch Block

Fig 154 –See legend on facing page

	Common Type of Right Bundle Branch Block	Variant Type of Right Bundle Branch Block
LEAD aVF	Completion of small terminal S wave or inscription of small secondary R wave	Completion of S wave
LEAD V	Downstroke of terminal R wave (The peak of the terminal R wave in lead V coincides ... val)	Completion of R wave or terminal R wave

Thus in summary the four electrocardiographic leads whose QRS deflections were described above and which can be considered to correspond to transverse vertical and anteroposterior leads of the vectorcardiogram record QRS complexes with the following types of configuration in right bundle branch block

	Common Type of Right Bundle Branch Block	Variant Type of Right Bundle Branch Block
LEAD I	qRs or qRS	Rs or RS or qRs
LEAD aVF	RSR rSr' qR Rs or RR	rS
LEAD V_1	rSR rsR or occasionally rR	Slurred R qR rSR or RR
LEAD V_6	Same as in lead I	RS or rS

VENTRICULAR REPOLARIZATION

Secondary changes in the S–T segments and the T waves usually accompany the altered QRS complexes in right bundle branch block Because of the delay in right ventricular depolarization the regions first activated (the free wall of the left ventricle and the septum which project negative repolarization potentials on right precordial leads for example) presumably begin to repolarize prior to the completion of the activation process in other areas of the heart principally the free wall of the right ventricle The direction of repolarization may also be reversed in the right ventricle itself if it is true as some believe that in right bundle branch block there is an abnormal spread of the activation wave through the right ventricular wall However the ventricular gradient concept provides that every change in the area of the QRS complex causes a corresponding *secondary* change in the area of the ST–T complex which is equal in magnitude but opposite in direction to the former Therefore the greater the *positive area of the* QRS complex in right precordial leads the more prominent will be the S–T segment depression and T wave inversion These several factors act to rotate the S–T and T vectors away from the terminal instantaneous QRS vectors and the corresponding mean vector for the terminal 0 04 second of the QRS interval In the absence of primary T wave changes such as those due to myocardial ischemia, the ventricular gradient remains normal Repolarization forces produced early in ventricular diastole sometimes depress the S–T segments in right precordial leads the T waves being inverted in these same leads Because the mean instantaneous T spatial vectors in right bundle branch block tend to be almost 180 discordant to the terminal mean instantaneous QRS spatial vectors not only are the T waves invariably inverted in leads V_1 and V_2 as indicated above but they are equally invariably *upright in leads* V_5 and V_6 often despite the presence of severe coexisting myocardial disease Thus if an upright T wave following an RSR *deflection is noted in lead* V_1 *then the diagnosis of* uncomplicated right bundle branch block should be questioned

VECTORCARDIOGRAPHIC FINDINGS

Just as in the preceding discussion of the instantaneous VA vectors and related electrocardiographic findings in the common and variant types of right bundle branch block the description of the vectorcardiographic findings in these two pattern variants will be presented *in parallel* in the following pages

	Common Type of Right Bundle Branch Block	*Variant Type of Right Bundle Branch Block*
LEAD aVF	Completion of upstroke of a small R wave	Completion of downstroke of a deep S wave
LEAD V_1	Nadir of S wave (following small initial R wave) which is of average or less than average depth in right bundle branch block	Nadir of relatively shallow S wave or the lowest point of the incisura separating two R waves When the 0 04 second VA vector is anteriorly placed it sometimes contributes to the upstroke of an R wave

0 06 SECOND VA VECTOR

As depolarization forces generated by the left ventricle begin to subside during the final 0 04–0 06 second of the QRS interval those produced by continued left to right septal activation become increasingly predominant This is reflected in the fact that the 0 06 second VA vector is directed to the right anteriorly and either more or less horizontally or in the case of the variant type of right bundle branch block pattern superiorly

	Common Type of Right Bundle Branch Block	*Variant Type of Right Bundle Branch Block*
	0 06 second VA vector directed to the right anteriorly and horizontally	0 06 second VA vector directed to the right anteriorly and superiorly
LEADS I AND V_6	Downstroke of a broad terminal S wave	Downstroke of a broad and usually relatively deep terminal S wave
LEAD aVF	Downstroke of R wave or beginning downstroke of shallow terminal S wave	Continued downstroke of deep S wave
LEAD V_1	Beginning upstroke of a secondary R wave which is wider and taller than the initial R wave	Upstroke or continued upstroke of an R wave

0 08 SECOND AND SUBSEQUENT VA VECTORS

Late in the QRS interval onset of right ventricular activation finally occurs the excitation impulse having reached the right ventricular myocardium either via the Purkinje fibers or by direct muscle fiber to muscle fiber spread from the left ventricle Whether the right ventricle depolarizes in a normal or abnormal manner the fact remains that right ventricular activation occurs so late in the QRS interval that unopposed electrical forces are in effect tacked on at the end of ventricular activation While the magnitude of these forces in the common type of right bundle branch block can be explained perhaps by the fact that they are unopposed this explanation is not applicable to the very large terminal QRS forces frequently observed in the variant pattern of right bundle branch block The magnitude of these forces is such as to make almost inescapable the conclusion that they reflect totally aberrant activation of the right ventricle The late instantaneous VA vectors lead to prolongation of the QRS interval to 0 11 second or longer and are directed toward the effective electrical site of the right ventricle—that is to the right anteriorly and horizontally in the case of the common type of right bundle branch block and either horizontally or slightly superiorly in the case of the variant pattern of right bundle branch block

	Common Type of Right Bundle Branch Block	*Variant Type of Right Bundle Branch Block*
	0 08 second and subsequent instantaneous VA vectors directed to the right anteriorly and horizontally	0 08 second and subsequent VA vectors directed either horizontally or slightly superiorly anteriorly and to the right
LEADS I AND V_6	Completion of relatively shallow terminal S wave	Completion of deep wide terminal S wave

	Common Type of Right Bundle Branch Block	Variant Type of Right Bundle Branch Block
LEAD aVF	Completion of small terminal S wave or inscription of small secondary R wave	Completion of S wave
LEAD V		Completion of R wave or terminal R wave

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Thus in summary the four electrocardiographic leads whose QRS deflections were described above and which can be considered to correspond to transverse vertical and anteroposterior leads of the vectorcardiogram record QRS complexes with the following types of configuration in right bundle branch block

	Common Type of Right Bundle Branch Block	Variant Type of Right Bundle Branch Block
LEAD I	qRs or qRS	Rs or RS or qRs
LEAD aVF	RSR rSr qR Rs or RR	rS
LEAD V	rSR rsR or occasionally rR	Slurred R qR rSR or RR
LEAD V₆	Same as in lead I	RS or rS

VENTRICULAR REPOLARIZATION

Secondary changes in the S–T segments and the T waves usually accompany the altered QRS complexes in right bundle branch block. Because of the delay in right ventricular depolarization the regions *first activated* (the free wall of the left ventricle and the septum which project negative *repolarization* potentials on right precordial leads for example) presumably *begin to repolarize* prior to the completion of the activation process in other areas of the heart, principally the free wall of the right ventricle. The direction of repolarization may also be reversed in the right ventricle *itself* if it is true as some believe that in right bundle branch block there is an abnormal *spread of the activation* wave through the right ventricular wall. However the ventricular gradient concept provides that every *change* in the area of the QRS complex causes a *corresponding* secondary *change in the area of the ST–T complex which is equal in magnitude* but opposite in direction to the former. Therefore the greater the positive area of the QRS complex *in right precordial* leads the more prominent *will be the S–T* segment depression and T wave inversion. These several factors act *to rotate the S–T and T vectors* away from the terminal instantaneous *QRS* vectors and the corresponding mean vector for the terminal 0 04 second of the QRS interval. *In the absence* of primary T wave changes such as *those* due to myocardial ischemia the ventricular gradient remains normal. Repolarization forces produced early in ventricular diastole sometimes depress the *S–T segments in* right precordial leads the T waves being inverted in *these same leads*. Because the mean *instantaneous* T spatial vectors in right bun*dle branch block* tend to be almost 180 *discordant to the terminal mean instantaneous QRS spatial* vectors *not only* are the T waves invariably inverted in

[illegible]

disease. *Thus if* an upright T wave following an RSR deflection is noted in lead V₁ then the diagnosis of *uncomplicated right bundle* branch block should be questioned

VECTORCARDIOGRAPHIC FINDINGS

Just as in the preceding discussion of the instantaneous VA vectors and related electrocardiographic findings in the common and variant types of right bundle branch block *the description of the vector cardiographic findings in these two pattern variants will be* presented in parallel *in the following* pages

COMMON TYPE OF RIGHT BUNDLE BRANCH BLOCK

VARIANT TYPE OF RIGHT BUNDLE BRANCH BLOCK

Horizontal QRS Loop

1 Initial deflection written to the right and anteriorly just as normally

1 Although the initial deflection of the horizontal QRS loop in this (variant) pattern is frequently written to the right and anteriorly in almost as many instances the horizontal QRS loop immediately moves to the left and anteriorly or less commonly to the left and slightly posteriorly (without electrocardiographic or clinical evidence of infarction)

2 The major part of the horizontal loop is usually written in a counterclockwise direction (rarely in a clockwise direction) to the left and at first anteriorly and later posteriorly (rarely is the afferent limb of the loop situated anterior to the efferent limb) The average orientation of the maximal leftward mean instantaneous QRS vector in our cases was at 0 in the horizontal reference frame

2 In this pattern the leftward portion of the horizontal loop is far more variable in appearance than in the common pattern Thus the maximal leftward mean instantaneous QRS vector may not extend as far to the left as in the common type of right bundle branch block however the average orientation of this vector is 0 like that in the common pattern Often this part of the loop is entirely clockwise inscribed or presents a figure-of-eight configuration with the proximal component counterclockwise inscribed and the distal component clockwise inscribed

3 In both patterns (common and variant) the afferent limb of the horizontal QRS loop does not return to the point of origin but continues in a rightward and predominantly anterior direction to inscribe a terminal finger like appendage whose average orientation in the two patterns was exactly the same in our cases—i e along the +120 axis of the horizontal reference frame On the whole the terminal appendage to the QRS loop in the variant type of right bundle branch block differed from that in the common type in two respects first the maximal mean instantaneous vector of the appendage tended to be relatively quite large compared to the preceding leftward portion of the horizontal loop and second the conduction delay as evidenced by the close spacing of the time dashes was more prominent

Right Sagittal QRS Loop

1 The sagittal QRS loop generally consists of two well delineated components Of these the first inscribed resembles to some extent the normal sagittal QRS loop in that it is written in a clockwise direction anteriorly and inferiorly and then returns posteriorly toward the electrical null point The maximal mean instantaneous QRS vector of this part of the sagittal loop lies on the average at +105 in the sagittal reference frame

1 The sagittal QRS loop in the variant pattern of right bundle branch block does not exhibit the two com

after it turns in a clockwise direction superiorly and posteriorly or slightly anteriorly The maximal mean instantaneous QRS vector for the first 0 04 second of the QRS interval was oriented on the average at −80 in our cases

2 Instead of returning to the electrical null point the afferent limb of the sagittal loop turns anteriorly and inscribes a long finger like appendage directed almost horizontally anteriorly Conduction delay is present in the terminal appendage more often than not but the delay is not usually striking The average orientation of the maximal anterior mean instantaneous QRS vector tends to be about +5

2 After the efferent limb of the sagittal loop has reached its maximally superior extent it turns anteriorly in a clockwise direction and descends gradually The maximal anterior mean instantaneous QRS vector in this pattern is usually directed essentially horizontally its average orientation being at about −10 Conduction delay in the terminal anterior part of the sagittal loop is ordinarily more prominent than in the sagittal loop of the common pattern

Frontal QRS Loop

1 As a general rule the frontal QRS loop in the common type of right bundle branch block presents either of the following appearances

a) The loop may be entirely clockwise inscribed despite a relatively horizontal orientation of its long axis

1 The frontal loop is usually written initially inferiorly and to the right and/or to the left but very quickly it moves superiorly The remainder of the loop is inscribed in a counterclockwise direction superiorly and to the left and then superiorly and to the right the latter segment of the loop displaying marked conduction delay

COMMON TYPE OF RIGHT BUNDLE BRANCH BLOCK

The terminal part of the loop showing conduction delay is situated inferiorly and to the right

h n o e or less

fi
fe
w
is clockwise inscribed to the right and u

taneous vector was +165

VARIANT TYPE OF RIGHT BUNDLE BRANCH BLOCK

Occasionally the frontal loop is inscribed superiorly and to the right or left initially then inferiorly and to the left and finally superiorly and to the right

2 The average orientation of the maximal leftward mean instantaneous QRS vector in our cases was −10 that of the maximal rightward instantaneous vector −160

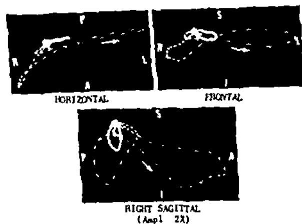
T sE Loop

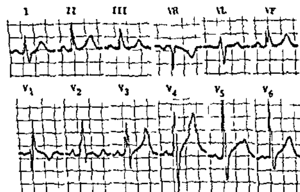
The most consistent feature of the T sE loop in right bundle branch block is that it is always directed away from the terminal vectors of the QRS sE loop

S–T Vector

In
S–T vector n

plexes in the electrocardiogram and the corresponding portion of the QRS sE loop of the vectorcardiogram appear essentially normal The prominent terminal deflection of the QRS sE loop to the right anteriorly and slightly inferiorly is diagnostic of right bundle branch block and is related to the electrocardiographic findings in leads V and V The T sE loop of the vectorcardiogram tends to parallel the long axis of the QRS sE loop but is directed away from the terminal deflection of the loop Thus leads I and V record upright T waves while lead V_1 registers an inverted T wave





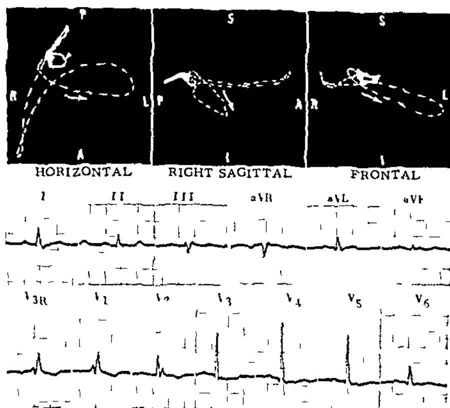
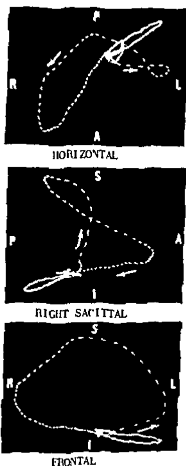


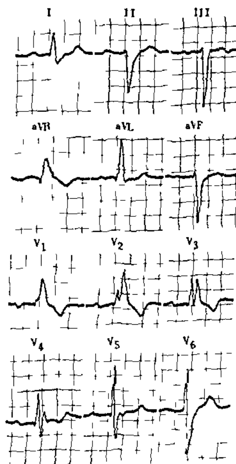
Fig 156 —Electrocardiographic and vectorcardiographic findings in the common type of right bundle branch block Note in the electrocardiogram that lead V re cords only a minute terminal S wave Moreover the S waves of the RSR deflections in leads V_{3R} and V_1 barely extend below the isoelectric base line The explanation for these findings is evident in the vectorcardiogram Thus the first half of the QRS sÊ loop is situated anteriorly and this is responsible for the diminutive S waves in the right precordial leads In addition the terminal deflection of the QRS sÊ loop is directed almost straight anteriorly and only slightly to the right Consequently the mean instantaneous QRS spatial vectors of the terminal part of the QRS sÊ loop project maximally on the axes of leads V_{3R} and V_1 and minimally on the negative half of the axis of lead V The terminal deflection of the QRS sÊ loop in this figure as well as in Figure 155 displays no evidence of localized conduction delay

Fig 157 —Electrocardiographic and vectorcardiographic findings in the variant type of right bundle branch block

In the electrocardiogram the QRS interval is 0 18 second there is marked left axis deviation of A QRS lead V_1 displays a slurred wide R wave and lead V_6 registers an RS deflection the terminal S wave being both deep and wide Note the absence of a normal septal Q wave in lead V

The vectorcardiographic QRS sÊ loop in the variant right bundle branch block pattern differs from that in the common type of right bundle branch block as follows there is an abnormal initial deflection of the loop to the left anteriorly and slightly inferiorly the sagittal and frontal QRS loops are inscribed entirely superiorly the latter usually having a counterclockwise inscription the first part of the horizontal QRS loop written to the left is dwarfed by the large rightward and anterior terminal deflection subsequently inscribed and the terminal limb of the QRS sÊ loop shows conduction delay



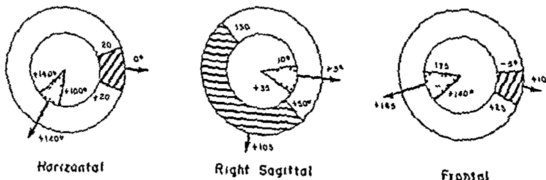


Grishman and his associates have described a pattern which is similar to the variant pattern of right bundle branch block discussed above with this one *and possibly* all important distinction in the QRS sE loop pattern of Grishman and his co workers the sagittal and frontal QRS loops are situated markedly superiorly and the terminal mean instantaneous vectors are not directed to any significant degree anteriorly *nor do* they form a typical terminal rightward and anterior appendage in the horizontal projection (*Fig 153*) These investigators believe the pattern to be the equivalent of that designated "left ventricular hypertrophy with terminal conduction delay" in Chapter 16 They point out that since the terminal QRS forces are directed markedly superiorly in "left ventricular hypertrophy with terminal conduction de-lay" minor differences in the position of these forces with respect to the anteroposterior (Zc) lead may result in the *terminal portion of the QRS sE loop* being inscribed anteriorly or posteriorly If there is an anterior return of the loop the electrocardiogram pre- ... f right bundle branch block

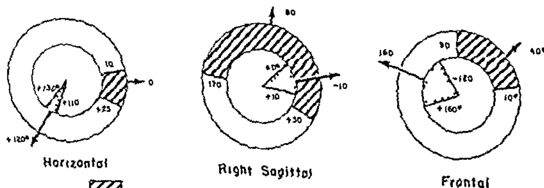
... terminal S waves appears in the precordial ...

None of the QRS sE loop patterns described so far in this chapter have been observed by the authors of this text to produce electrocardiographic findings of the type ascribed to classic right bundle branch block This entity which was described at a time when only scalar electrocardiograms were available *is said to* present the following features

Common Type of Right Bundle Branch Block



Variant Type of Right Bundle Branch Block



MAXIMAL INITIAL .04 SEC MEAN QRS VECTOR

MAXIMAL TERMINAL .04 SEC MEAN QRS VECTOR

e
variant

1. Lead I shows a small narrow R wave and a deeper and wider S wave
2. Leads II and III usually show narrow R waves taller than in lead I which may be preceded by a small Q wave and followed either by a second upright deflection or a smaller S wave
3. Leads V_{3R} to V_2 record initial small Q waves and terminal large slurred and widened R waves while from right to left across the precordium RS deflections are recorded with diminishing R wave *amplitude and increasing depth of the* S wave

As a result of their studies Grishman and his asso ciates have concluded that in many instances th pattern of classic right bundle *branch block may b* due primarily to marked changes in the balance c electrical forces as the result of infarction involvin the lateral posterior and inferior aspects of the lef *ventricle Richman and Wolff* have observed patient whose electrocardiograms were consistent with th diagnosis of classic right bundle branch block bu whose vectorcardiograms were indicative of left bun dle branch block and extensive septal and inferolat eral myocardial infarction

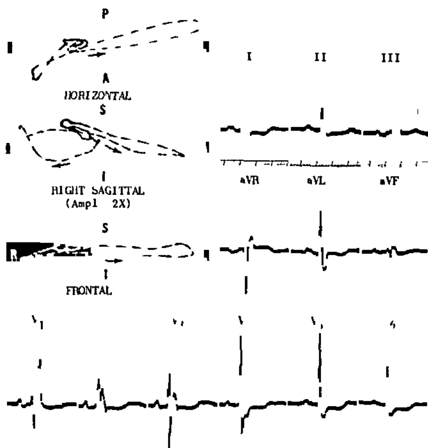


Fig 159 — Electrocardiographic

ing left ventricular hypertrophy are left axis deviation of A QRS in the frontal plane tall R waves depressed S–T segments and diphasic T waves in leads I aVL V_5 and V_6 and the relatively deep S wave of the RSR deflection in lead V In the vector cardiogram the findings suggestive of coexisting left ventricular hyper trophy are increased magnitude of the leftward instantaneous vectors of *the QRS sÊ loop posterior and supe* rior rotation of the long axis of the

terminus of the *QRS sÊ loop)* and the small T sÊ loop The vectorcar diographic findings indicative of right bundle branch block are obvi ous and will not be described

DIAGNOSIS OF COEXISTING VENTRICULAR HYPERTROPHY

The following electrocardiographic findings have been proposed by Barker and Valencia as suggestive of right bundle branch block plus right ventricular *hypertrophy*

1. If the secondary R wave in V_1 exceeds 10 mm in amplitude in incomplete right bundle branch block and 15 mm in complete right bundle branch block right ventricular hypertrophy may be present
2. The R and R waves registered in right precordial leads should be clearly separated by the S wave
3. The R waves recorded in left precordial leads are relatively small in comparison with the increased size of the S waves

The general experience has been *that the above* criteria are not of great value in detecting right ventricular hypertrophy in the presence of right bundle branch block When we applied these criteria to the electrocardiograms of patients with *mitral stenosis* showing RSR deflections in lead V_1 we found that the diagnosis of right ventricular hypertrophy could not be made in a single case even though the vector

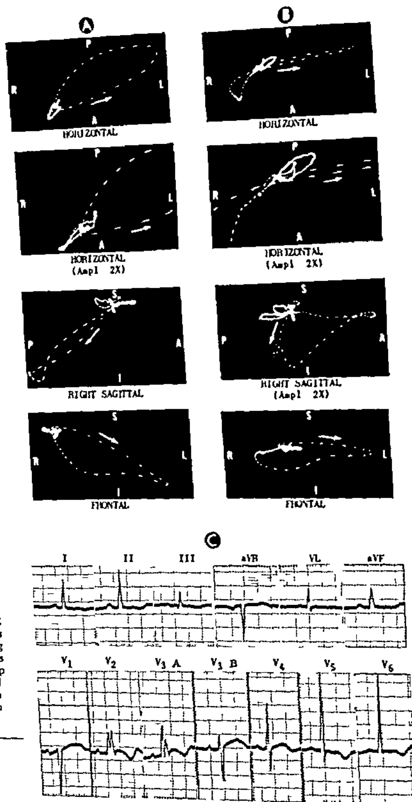


Fig 160 —Electrocardiographic and vectorcardiographic findings in intermittent right bundle branch block and left ventricular hypertrophy

The electrocardiogram **(C)** is diagnostic of left ventricular hypertrophy however in leads V and V "A" the QRS complexes have widened to 0 14 second and consist of notched R waves with small terminal S waves In lead V B the intraventricular block has disappeared again The appearance of the widened ventricular complexes in leads V and V "A" suggests right bundle branch block which was verified by later electrocardiograms (not shown)

The two sets of vectorcardiographic loops **(A** and **B)** were obtained during the recording of a single vectorcardiogram from the patient whose electrocardiogram was just described The planar QRS loops in **A** present the features of left ventricular hypertrophy In **B** note that, despite the appearance of right bundle branch block the planar QRS loops show little change from those in **A** for about the first 0 03 second, but terminally a rightward and anterior deflection of the loop is written which shows closely spaced time markings The vectorcardiogram is therefore diagnostic of right bundle branch block Note also that the T sÊ loop in the vectorcardiogram in **A** is directed almost 180 away from the QRS sÊ loop while the T sÊ loop in **B** is essentially concordant to the long axis of the QRS sÊ loop This secondary change in T sÊ loop orientation reflects solely the alteration in ventricular depolarization consequent to the right bundle branch block.

cardiograms showed typical right ventricular hypertrophy patterns

Braunwald and his associates have observed several instances in which vectorcardiographic loops diagnostic of right ventricular hypertrophy showed marked conduction delay in their terminal portions probably due to right bundle branch block. These investigators were able to demonstrate in these cases a delayed onset of mechanical right ventricular systole such as is found in right bundle branch block but not in right ventricular hypertrophy alone. Burch and his associates and Wolff and his co-workers have also described combined right ventricular hypertrophy and right bundle branch block as producing displacement anteriorly and to the right of a QRS sÊ loop otherwise typical of right bundle branch block.

Grishman and Scherlis have observed in some cases of combined left ventricular hypertrophy and right bundle branch block that the QRS loop is oriented more superiorly than is usually the case with right bundle branch block alone. This is in general agreement with our findings and those of Burch and his associates (Figs 159–160).

OTHER TYPES OF INTRAVENTRICULAR BLOCK

Focal or Peri-infarction Intraventricular Block

This type of intraventricular block is thought to occur primarily as a complication of myocardial infarction. Accordingly, discussion of it will be deferred until later (Chapter 21).

Diffuse Intraventricular Block

Very rarely a marked intraventricular block without an associated complete interruption of the left bundle branch may result from left ventricular hypertrophy and dilatation, diffuse myocardial fibrosis, the use of quinidine or hyperkalemia. Apparently there is merely a diffuse slowing of conduction through the ventricular wall. Although the electrocardiographic pattern resembles that of left bundle branch block in some respects, the ability of the interventricular septum to depolarize from left to right usually produces normal Q waves in left precordial leads.

The vectorcardiographic counterpart of this type of intraventricular block has not been described. In fact, we studied vectorcardiographically only 1 case of diffuse intraventricular block. In this instance the heart at postmortem examination was found to have endocardial fibroelastosis and marked ventricular

t

zarre appearance, while the vectorcardiogram displayed the following findings: (1) marked conduction delay in the efferent limbs of the horizontal and frontal QRS loops and in the afferent limb of the sagittal loop; (2) abnormal initial deflection of the QRS sÊ loop to the left and posteriorly; (3) horizontal QRS loop entirely clockwise inscribed to the left and posteriorly; (4) frontal QRS loop almost linear and written almost directly to the left; and (5) T sÊ loop 180 discordant to the long axis of the QRS sÊ loop.

Myocardial Ischemia, Injury, and Infarction
General Considerations

18

EXPERIMENTAL OCCLUSION of the coronary artery in dogs produces electrocardiographic changes which correlate in a general way *with the severity* and reversibility of the histologic alterations in the myocardium. These electrocardiographic changes appear in three stages of increasing abnormality—namely ischemia, injury, and necrosis. The ischemia and injury phases are not accompanied by *irreversible* alterations in the muscle cells, but once necrosis has supervened, the damage to the muscle fibers becomes *irreversible*. In addition to the reduction in blood supply to the affected myocardium, other noxious influences *sometimes* produce the histologic and electrocardiographic changes of ischemia, injury, and necrosis. However, in the following chapters the discussion will center primarily on ischemia, injury, and infarction secondary to reduced or absent blood supply to the involved myocardium, although the principles developed apply equally well to other conditions such as myocarditis, pericarditis, etc. (see Chapter 21).

ISCHEMIA

The distribution of the coronary arteries is such that normally the blood supply of the outer layers of ventricular musculature is relatively greater than that of the deeper subendocardial layers. This disparity in blood supply may be enhanced by the transmission of greater degrees of left intraventricular pressure to the subendocardium than to the outer layers of myocardium. The high pressure tends to collapse the blood vessels within the subendocardium. As a consequence of the foregoing two factors, myocardial ischemia appears first, and is more extensive, in the subendocardium.

Subendocardial Ischemia

Ischemia leads to a local delay in the onset of recovery and therefore to a local prolongation of the *excited state*. For reasons previously stated, ischemia secondary to diminished regional blood supply begins in the subendocardial layer of muscle. Since repolarization normally proceeds in an epicardial-to-endocardial direction, *delayed recovery in the subendocardial* muscle does not reverse the direction of repolarization but merely lengthens its duration locally (thereby prolonging the Q–T interval). Thus the ischemic subendocardial muscle continues to repolarize for a time after opposing repolarization potentials from other regions have begun to subside. This circumstance tends to *rotate the late instantaneous T vectors toward* the effective site of the ischemia and to increase the magnitude of these vectors, since they are relatively unopposed. Consequently, leads over the involved myocardium record upright T waves of increased amplitude and duration.

Transmural (Epicardial) Ischemia

When ischemia extends transmurally to the epicardium, it apparently has a more profound effect on the recovery of epicardial cells than it has on recovery of *endocardial* cells. In the ischemic myocardium, onset of repolarization is delayed longer in the epicardium

than in the endocardium, with the result that the endocardial muscle fibers are the first to recover. The wave of repolarization then spreads through the involved muscle wall in an endocardial to epicardial direction, the reverse of normal. This local reversal in the direction of repolarization causes the repolarization forces generated by the involved myocardium to be directed just the opposite of normal. For example, at each instant during ventricular repolarization, transmural ischemia of the anterior wall of the left ventricle gives rise to T vectors directed posteriorly which, added vectorially to the normal T vectors originating in unaffected portions of the left ventricle, yield mean instantaneous T vectors directed more or less away from the effective site of the ischemia. These instantaneous T vectors project on the negative portions of the axes of leads overlying the region of ischemia so that these leads record inverted T waves. Since ventricular depolarization and therefore the orientation of the mean instantaneous QRS vectors remain undisturbed by myocardial ischemia, the displacement of the instantaneous T vectors usually results in an increased angular divergence of the mean QRS and T vectors. If the QRS–T angle becomes sufficiently wide, leads registering upright QRS complexes may record inverted T waves, and vice versa. Inasmuch as transmural myocardial ischemia causes a local prolongation in the duration of the excited state, it is associated with an abnormal ventricular gradient. For this reason, the T wave abnormalities of ischemia are primary in type.

The vectorcardiographic T sÊ loop has not been studied extensively as yet, and most of the available information pertains to its orientation with respect to the QRS sÊ loop. In transmural myocardial ischemia, the T sÊ loop tends to rotate away from the affected portion of the myocardium, and thus the angular deviation between the QRS sÊ and T sÊ loops usually but not invariably becomes abnormally increased (normally, the angle of deviation rarely exceeds 45°). An abnormal angle of deviation between the QRS sÊ and T sÊ loops may appear before T wave abnormalities become evident in the scalar electrocardiogram, since a relatively marked degree of angular deviation is generally required to produce abnormal T waves. The efferent limb of the normal T sÊ loop, like the upstroke of the normal T wave, is inscribed more slowly than the afferent limb of the loop and the downstroke of the T wave. Large elongated T loops have been described in acute myocardial infarction while old infarctions have been associated by some investigators with the presence of small round T loops. These variations in T loop configuration and inscription are not fully understood at present and require further study.

INJURY

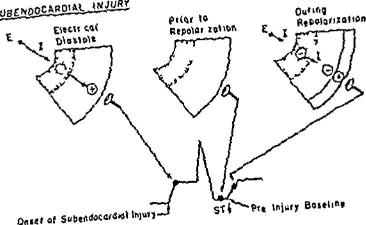
Diastolic Current of Injury

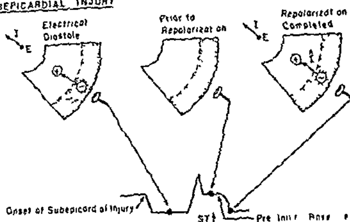
Injury of a resting myocardial cell has two effects: (1) it decreases the degree of polarization so that there are fewer positive charges per unit surface area, and (2) it abolishes the selective permeability of the injured cell membrane so that negative ions are permitted to escape outside the cell, where they are neutralized by positive ions. As a result, during electrical diastole the injured membrane (Fig. 161) comes to be negatively charged relative to the intensely polarized and positively charged adjacent membrane, and a potential difference exists between the two regions. If the myocardial cell is situated in a volume conductor, a diastolic current of injury flows from the region of greater potential to the region of lesser positive or more negative potential—that is, from the outer to the inner surface of the cell membrane through the region of injury. The electrical field which is created in the surrounding conducting medium during electrical diastole can be represented by a vector directed from the injured area toward the intact cell membrane. This vector, hereafter labeled *I*, depicts the electrical field of the diastolic current of injury, while a second vector, –*I*, discussed later (pp. 258 and 259), represents the field of the systolic current of injury.

If it were possible to place an electrocardiographic lead electrode over the injured end of the resting muscle cell, the base line of the tracing would be deflected downward at the time of injury because *I* is directed away from the recording electrode. On the other hand, the precise instant at which the diastolic current of injury appears and depresses the electrocardiographic base line is never observed in clinical electrocardiography. Consequently, the direct effect of the diastolic current of injury on the electrocardiogram is never evident and will be ignored hereafter in this text.

Systolic Current of Injury

During electrical systole, a current of injury flows toward the injured cell membrane and therefore in a





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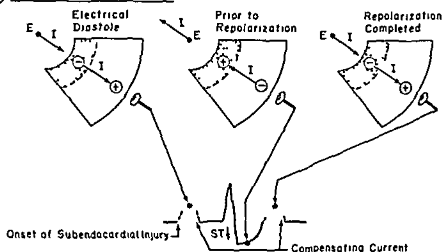
direction just the opposite of that of the diastolic current of injury The *mechanism responsible for this* systolic current of injury has been the subject of some dispute The conflicting explanations which have been offered can be reduced for the sake of simplicity to the two mechanisms presented below (*a*) disappearance of the diastolic current of injury and (*b*) blocking of depolarization

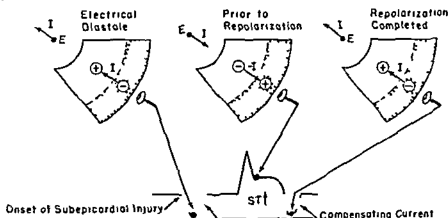
Disappearance of the diastolic current of injury

(Fig 181) —As noted before the diastolic current of *injury is* present only *during* ... hat ... on ... com ... injury ... corded ... *aspect of the cell ascends to its original* preinjury level The elevated S-T segment registered *in* the electrocardiogram and the apparent pres

ence of a systolic current of injury therefore actually reflect the absence of the diastolic injury vector according to the proponents of this concept Repolarization of the cell restores the potential difference between injured and intact membrane and so following inscription of the T wave the base line of the tracing descends once again

Blocking of depolarization (Fig 162) —This concept is based on the premise that there is partial or complete blocking of the depolarization wave at the borders of the region of injury Thus even though the *injured* membrane of the resting cell has fewer positive charges per unit surface area than adjacent intact *membrane* with the completion of depolarization the injured membrane (where the activation wave has been completely or partially blocked) retains its positive charges and becomes positively charged with respect to the neutralized membrane surfaces As a result a systolic current of injury flows toward the injured cell membrane during the interval between





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However it is postulated that the depolarization wave is blocked at the boundaries of the injured region so that the latter retains some of its dipoles at a time when uninjured muscle elsewhere has been completely discharged Therefore before repolarization a systolic current of injury appears (represented by the vector $-I$) and projects negative voltage or a depressed S–T segment on the recording lead With onset of repolarization the diastolic current of injury again appears and it is immediately neutralized by the compensating current In subepicardial injury (B) the systolic current of injury vector ($-I$) is directed toward the recording electrode so that the S–T segment of the electrocardiogram is displaced upward As the explanation for the S–T segment deviations observed clinically and experimentally in myocardial injury the blocking of depolarization theory of the systolic current of injury is in general more widely accepted than the disappearance of the diastolic injury concept

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Subendocardial and Subepicardial Injury

What has been indicated previously concerning the effects of injury of a single heart muscle cell holds true for injury of a region of the ventricular myocardium When the injury involves the subendocardial layer of ventricular muscle the activation wave can be considered to be blocked at the borders of the injured muscle At the completion of electrical systole the subendocardial muscle therefore retains some of its positive charges while outer muscle layers of the involved ventricular wall are completely depolarized A potential difference exists between positively charged subendocardial muscle and relatively negatively charged epicardial muscle and a systolic current of injury flows toward the injured muscle layer The electrical field of the injury current resulting from subendocardial injury can be represented by an −I vector originating at the electrical center of the heart and directed away from the effective site of the injured muscle Thus the −I vector of subendocardial injury projects on the negative halves of the axes of leads overlying the region of the injury with the result that these leads display depressed S–T segments

When injury is limited to subepicardial muscle the latter is positively charged (for the same reasons as described above) and the subendocardial muscle relatively negatively charged at the end of electrical systole In this instance the systolic injury vector (−I) is directed from the electrical center of the heart toward the effective site of the injured muscle and therefore projects elevated S–T segments on leads overlying the region of injury

The −I or S–T Vector of the Vectorcardiogram

If there is a significant systolic current of injury the QRS sE loop of the vectorcardiogram does not after its inscription return to its point of origin because a potential difference continues to exist between

and direction can be depicted by a vector arrow drawn from the origin of the QRS sE loop to its terminus In anterior subepicardial injury this S–T vector is usually directed to the left inferiorly and to a varying degree anteriorly in subendocardial injury to the right posteriorly and superiorly

The T sE loop is inscribed from the termination of the open QRS sE loop to the point of origin

INFARCTION

Infarction is the term used to designate necrosis of heart muscle secondary to a deficient blood supply Clinical myocardial infarction occurs primarily as a complication of coronary artery disease (its immediate cause usually being coronary thrombosis) and can be considered the end stage of the ischemia injury necrosis sequence described earlier in this chapter Most infarctions are found on pathologic study to be situated in the left ventricular free wall and/or interventricular septum The only exceptions to this rule are anteroseptal infarctions (which may involve a small strip of adjacent right ventricular wall) atrial infarctions and right ventricular infarctions although the latter two types of infarction are recognized relatively rarely Atrial infarction produces *a shift of the P–Ta segment* but this effect is usually obscured by the larger ventricular deflections appearing at about the same time Difficult as it may be to recognize an atrial infarction it is virtually impossible to establish the diagnosis of right ventricular infarction This is not surprising if one recalls that the right ventricle normally contributes only a quarter of the activation potentials producing the QRS complex Thus the disappearance of some or all of these right ventricular forces as the result of infarction would not in most instances be likely to alter the QRS configuration significantly

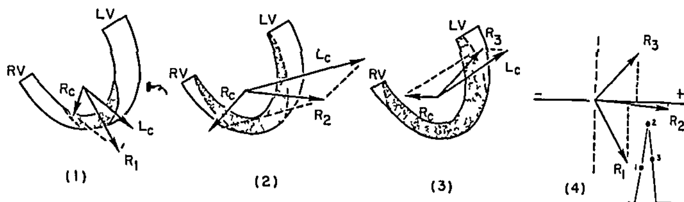


Fig 163 —Genesis of the diagnostic Q waves and related QRS abnormalities in myocardial infarction **A** normal heart The ventricular portion of the heart is depicted schematically but with the omission of the septum The activation wave front in each ventricle the component vector representing the electrical force arising in each ventricle and the resultant vector of the two component vectors are presented as they exist at three different stages in the activation process In **A** (4) the three resultant or mean instantaneous vectors have been projected on the axis of derivation of a transverse lead and the values obtained have been used to construct the ventricular complex recorded by the lead **B** myocardial infarct (solid black) The electrically inert infarcted muscle fails to produce component vectors L in stages 1 and 2 of the activation process Consequently the right ventricular component vectors are unopposed and are equivalent to the resultant vectors R_1 and R R is unchanged As demonstrated in the lower two figures in **B** one can also determine the postinfarction resultant vector from the preinfarction vector since the former is the resultant of the latter and the

Mechanism of the QRS Abnormalities of Infarction

A deep wide Q wave appearing in a lead not normally recording Q waves of such prominence is perhaps the most typical QRS abnormality produced by infarction. Infarction Q waves were once ascribed to the transmission of negative cavity potentials through a "window" of necrotic myocardium to an overlying lead electrode. However, this explanation, like all others based on the localized potentials (or semidirect lead) concept of electrocardiography, is probably not valid when applied to body surface potentials. The mechanism outlined in the following paragraphs has as its basis the equivalent dipole or resultant vector concept, the validity of which seems for the most part established.

According to the theory currently accepted by most authorities, infarcted muscle may be considered electrically inert (Fig 163 A and B). Therefore, the electrical effect of a transmural infarction equals the electrical forces normally produced by the infarcted muscle at a given instant of the QRS interval subtracted from the balance of cardiac forces normally existing at the *same instant*. Thus, from a given preinfarction mean instantaneous vector and from the vector representing the *forces normally produced* by the infarcted region of the ventricle (the infarction vector), it should be possible theoretically to calculate the corresponding postinfarction mean instantaneous vector (Fig 164). This can be done simply by laying off the *two* vectors from the same point and then drawing a third vector, the postinfarction mean instantaneous vector, from the tip of the infarction vector to the tip of the preinfarction mean instantaneous vector. Since clinically the p e and postinfarction mean instantaneous vectors can be obtained from the vectorcardiogram or can be determined approximately from the electrocardiogram, it is also possible to calculate, by vector subtraction, the orientation of the infarction vector. In this instance the two mean instantaneous vectors are drawn from the same point and the infarction vector is then drawn from the tip of the postinfarction vector to the tip of the preinfarction vector.

Since the infarction vector represents the electrical forces normally generated by the infarcted ventricular muscle, it necessarily points toward the effective electrical site of the infarction. As previously indicated, the electrical field of the heart at a given instant, or the corresponding mean instantaneous QRS spatial vector, is determined by the balance arrived at between opposing electrical forces acting simultane

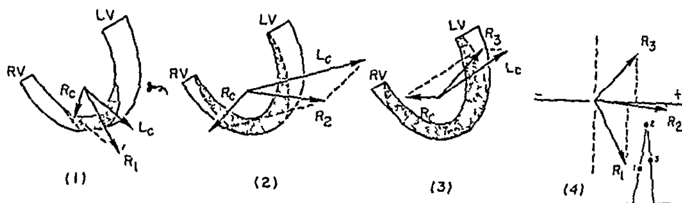
unbalanced forces of equal mag opposite direction. These unbalanced forces can be represented by an infarction vector like that described in the preceding paragraph but directed oppositely— he effective electrical site of the tha inf

on y the postinfarction direction and magnitude the postinfarction mean instantaneous vector, the latter preinfarction mean instantaneous vector and the infarction vector are plotted from the same point of origin and lines are then drawn parallel to the two vectors to form a parallelogram. The diagonal from the point of origin of the two component vectors to the intersection of the two lines completing the m the resultant of the infarction vector

stantaneous vector. It is obvious that the p tion instantaneous vector, like its component infarction vector, is shifted away from the effective electrical site of the infarction. For example, in anterior myocardial infarction the 0.02 second mean instantaneous vector tends to be shifted posteriorly, whereas normally it usually lies somewhat anterior. As a consequence, lead V which before onset of the infarction

instan jects By way of the left ventricle, the 0.02 second mean instantaneous vector may be displaced more anteriorly than normal. In this event lead V_2 may register an abnormally tall R wave. The QRS abnormality in either type of infarction clearly reflects an over all disturbance in the balance of cardiac forces and is not a manifestation of altered localized potentials.

It is evident from the points developed in the preceding paragraphs that the direction and magnitude of a given postinfarction mean instantaneous vector and, consequently, the electrocardiographic and vectorcardiographic changes produced by an infarction are functions of the following two variables: (1) the direction of the infarction vector (or the effective electrical site of the infarction) and (2) the orientation and magnitude of the preinfarction mean instantaneous QRS vectors. For example, the infarction vector in infarction of the anterior wall of the left ventricle is directed posteriorly. If the preinfarction mean instantaneous vectors during the initial 0.04 second of



B MYOCARDIAL INFARCTION

LV RV $R_c = R'_1$ (1)

LV RV $R_c = R'_2$ (2)

LV RV R_3 L_c R_c (3)

R_3 R'_2 R_1 − + (4)

LV RV $L_c = IV$ R_1 R_1 (1)

LV RV $IV = L_c$ R_2 R_2 (2)

R_c = Rt Ventricular Component Vector

L_c = Lt Ventricular Component Vector

$R_{1,2,3}$ = Successive Resultant Vectors

IV = Infarction Vector

$R'_{1,2}$ = Post-Infarction Resultant Vectors

Activated Muscle

Infarcted Muscle

front in each ventricle the component vector representing the electrical force arising in each ventricle ... ant vector of the two component vectors are presented as they exist at three different stages in the activation process In A (4) the three resultant or mean instantaneous vectors have been projected on the axis of derivation of a transverse lead and the values obtained have been used to construct the ventricular complex recorded by the lead B myocardial infarct (solid black) The electrically inert infarcted muscle fails to produce component vectors L in stages 1 and 2 of the activation process Consequently the right ventricular component vectors are unopposed and are equivalent to the resultant vectors R_1 and R_2 R is unchanged As demonstrated in the lower two figures in B one can also determine the postinfarction resultant vector from the preinfarction vector since the former is the resultant of the latter and the ... identical with the left ventricular component vectors in stages 1 and 2

the QRS interval happen to be directed superiorly the resultant of each of these instantaneous vectors ... of the infarction vector is a postinfarction instan ... n more

...

II III and aVF The electrocardiogram ... case is usually interpreted as showing both anterior and inferior or diaphragmatic infarctions whereas in ... the effective electrical site of the infarction is

...

differences in the direction of the infarction vector or in other words from differences in the electrical location of the infarct For this reason the classification of myocardial infarction utilized in this text is based on the effective electrical site of the infarction or the direction of the infarction vector

Criteria of Q Wave Abnormality

As a general rule the recognition of acute myocardial infarction in the electrocardiogram seldom presents too great a problem probably because in this *stage of infarction* the electrocardiogram displays not only QRS abnormalities but also characteristic shifts *in the S–T segments and* T wave *changes In contrast* it is quite difficult and sometimes impossible to distinguish the electrocardiographic residuals of a healed infarction from normal deflections This is particularly true in the case of infarction Q waves when they occur as late residuals in leads normally inscribing septal Q waves In evaluating Q waves to determine whether they represent an abnormality or not the following criteria are often quite useful

Lead I –Goldberger s criteria of abnormality are width of Q wave ≧ 0 04 second depth of Q wave = 1 mm or more provided the amplitude of the following R wave is approximately 5 mm According to Barker depth of Q wave ≧ 10% of the total QRS amplitude

Leads III and aVF –The criteria of Barker of Goldberger and of Myers are width of Q wave ≧ 0 01 second depth of Q wave > 25% of the amplitude of the following R wave

Lead aVL –Goldberger advocates that all of the following criteria be satisfied width of Q wave ≧ 0 04 second depth of Q wave > 50% of the amplitude of the following R wave presence of upright P and T waves in lead aVL and an rS deflection in lead aVR

Leads V_1 through V –Criteria of Sodi Pallares *depth* of Q wave > 3 mm (if Q wave is followed by a small R wave) prolonged *width of* Q wave *and* W shaped QRS deflection According to Goldberger width of Q wave ≧ 0 04 second *depth of Q* wave > 25% of amplitude of following R wave

Leads V_5 and V_6 –Barker s criteria width of Q wave ≧ 0 04 second depth of Q wave > 15% of total QRS amplitude

If in a given electrocardiogram there are Q waves in one or more leads satisfying the criteria listed above the Q waves can ordinarily be considered a reliable indication of the presence of *infarction* However the converse of this does not necessarily hold true that is the failure of Q waves to meet the criteria for abnormality certainly cannot be regarded as incontrovertible evidence against the diagnosis of infarction The application of the above criteria to the diagnosis of specific types of infarction and the limitations of the criteria in this regard are discussed later (p 291)

Evolution of an Infarction

It will be recalled that in experimentally produced myocardial infarction there appear in sequence the electrocardiographic changes of myocardial ischemia subepicardial injury and finally myocardial necrosis

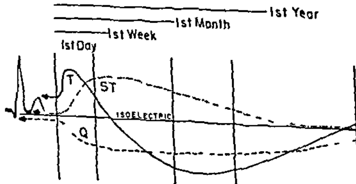
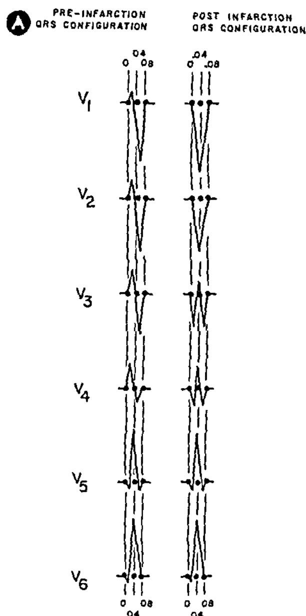
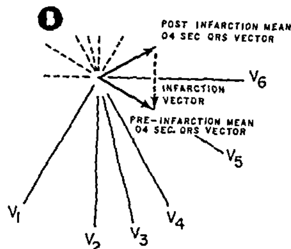
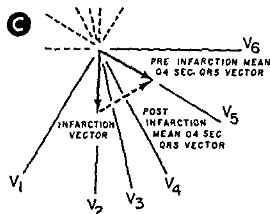


Fig 165 –Development and evolution of the S–T segment and the Q and T wave changes accompanying myocardial infarction as related to the time of onset of the infarction The more a curve rises above the isoelectric line the taller is the T wave or the greater the S–T segment elevation while the greater the descent of the curve below the isoelectric line the deeper is the Q wave or the inverted T wave (After Lepeschkin.)





Calculation of the Infarction Vector from the Pre-Infarction and Post-Infarction Mean 04 Sec QRS Vectors in Horizontal Plane



Calculation of the Post-Infarction Mean 04 Sec QRS Vector from the Corresponding Pre-Infarction QRS Vector and the Infarction Vector

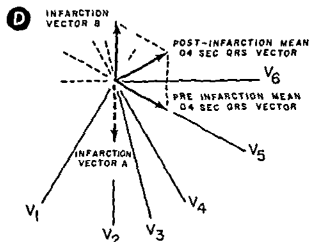


Fig 164 —**A–D** methods for calculating the infarction vector and the postinfarction mean instantaneous 0 04 second QRS vector It should be noted that strictly speaking the infarction vector as depicted in **B** and **C** is defined as the vector representing the forces normally produced by the infarcted area of ventricular muscle However as

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3 The QRS loop now distorted by the effect of the myocardial infarction tends to close as the S–T vector disappears The T loop becomes elongated and may be written at a constant rate of inscription The long axis of the T loop tends to be directed away from the area of infarction

In a strict sense the diagnosis of myocardial infarction can only be made if there is electrocardiographic evidence of muscle necrosis in the form of abnormal Q waves or equivalent QRS changes Nevertheless if there is a strong clinical suspicion of infarction S–T segment elevation of significant degree, although unaccompanied by QRS abnormalities should be considered suggestive evidence electrocardiographically of infarction until proved otherwise by serial tracings In most cases of infarction Q waves and deeply and symmetrically inverted T waves will have appeared in the diagnostic leads within the first 24–48 hours and the S–T segments will have begun their descent to the isoelectric base line Once S–T segment elevation has disappeared after an infarction it is no longer possible to date the infarction on the basis of the electrocardiographic findings in a single record since QRS and T wave abnormalities resembling those present in the acute phase of infarction can persist indefinitely

Terminology and Classification of Infarction Patterns

In the past the terminology of infarction tended to stress the anatomic location of the infarction (e g anterior posterior lateral) because the infarction s site was believed to govern almost entirely the distribution of QRS abnormalities in the electrocardiographic leads This method of designating the various infarction patterns was in accord with the classic electrocardiographic pathologic correlation studies of Myers However recent evidence and experience indicate that the electrical and anatomic locations of an infarction although often approximately the same frequently differ to a widely varying degree That discrepancies should occur between electrocardiographic and pathologic localization of infarctions is probably less surprising than is the fact that the two studies correlate as frequently as they do Two of the factors contributing to the inconsistencies in electrocardiographic and pathologic correlation are as follows

a) The electrocardiogram is recorded with distant indirect leads which respond to the cardiac potentials as if the latter were actually a single equivalent dipole This fact is not compatible with precise anatomic localization of a myocardial infarction

b) Undoubtedly factors other than the anatomic site of an infarction influence the distribution of QRS abnormalities in the electrocardiographic leads as was pointed out in the preceding discussion of the mechanism of the Q waves of infarction Thus the direction of the unbalanced forces produced by an infarction and perhaps even the time they appear in the QRS interval may well be modified by variations in intraventricular conduction perifocal block and/or coexisting abnormalities of ventricular muscle In view of the uncertainty as to the mechanism of the QRS abnormalities of infarction the meaning of these ab

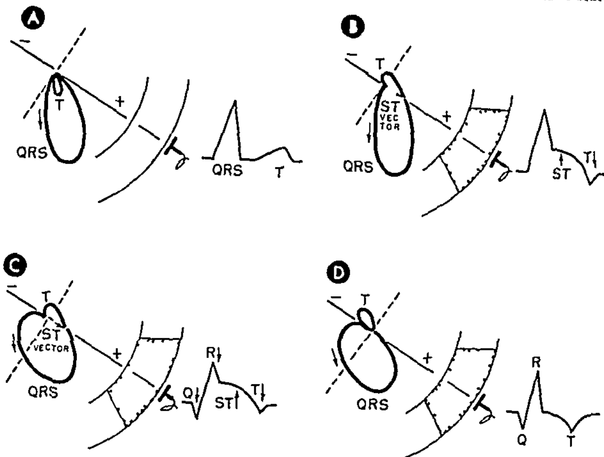


Fig 166 —Evolution of a myocardial infarction The related changes in the electrocardiogram and vectorcardiogram are depicted in A before infarction in B during the subepicardial injury and ischemia stage of infarction in C during the stage of myocardial necrosis or infarction and in D during regression of the injury pattern (See text for detailed description)

or infarction Clinically myocardial infarction passes through much the same sequence However the initial stage consisting of subendocardial ischemia soon followed by transmural *ischemia is ordinarily so transient* that the electrocardiographic abnormality first detected is the S–T segment elevation of subepicardial injury The evolution of electrocardiographic and related vectorcardiographic changes during a typical *myocardial infarction may be summarized in general* terms as follows (see also Figs 165 and 166)

ELECTROCARDIOGRAM	VECTORCARDIOGRAM
1 S–T SEGMENT ELEVATION appears in diagnostic leads and is usually the first indication of a developing infarction The T wave is often obscured by the S–T segment elevation but if it is discernible it may show slight terminal inversion The superimposition of S–T segment elevation on the pattern of an old infarction usually signifies fresh infarction in the region of previous involvement	1 This phase is reflected in the vectorcardiogram by failure of the QRS s£ loop to close The terminal portion of the loop does not return to its point of origin but tends to be displaced toward the effective site of subepicardial injury The S–T vector which is directed from the point of origin of the loop to its terminus points toward the area of injury The T loop may be small and directed away from the involved region

Duration This stage may last only a few hours However it usually begins to subside after a week and disappears completely within 2 weeks to several months Regression of the injury pattern heralds either the recovery or death of heart muscle Thus in the latter instance the descent of the S–T segments is accompanied by developing electrocardiographic evidence of myocardial necrosis

2 ABNORMAL Q OR QS WAVES usually make their appearance in diagnostic leads within several hours to sev	2 Because the electrically inert infarcted myocardium disrupts the normal balance of electrical forces the ini

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tion S–T segment elevation of significant degree although unaccompanied by QRS abnormalities should be considered suggestive evidence electrocardiographically of infarction until proved otherwise by serial tracings In most cases of infarction Q waves and deeply and symmetrically inverted T waves will have appeared in the diagnostic leads within the first 24–48 hours and the S–T segments will have begun their descent to the isoelectric base line Once S–T segment elevation has disappeared after an infarction it is no longer possible to date the infarction on the basis of the electrocardiographic findings in a single record since QRS and T wave abnormalities resembling those present in the acute phase of infarction can persist indefinitely

Terminology and Classification of Infarction Patterns

In the past the terminology of infarction tended to stress the anatomic location of the infarction (e g an erior posterior lateral) because the infarction s site was believed to govern almost entirely the distribution of *QRS* abnormalities in the electrocardiographic leads This method of designating the various infarction patterns was in accord with the classic electrocardiographic pathologic correlation studies of Myers However recent evidence and experience indicate that the electrical and anatomic locations of an infarction although often approximately the same frequently differ to a widely varying degree That discrepancies should occur between electrocardiographic and pathologic localization of infarctions is probably less surprising than is the fact that the two studies correlate as frequently as they do Two of the factors contributing to the inconsistencies in electrocardiographic and pathologic correlation are as follows

a) The electrocardiogram is recorded with distant indirect leads which respond to the cardiac potentials as if the latter were actually a single equivalent dipole This fact is not compatible with precise anatomic localization of a myocardial infarction

b) Undoubtedly factors other than the anatomic site of an infarction influence the distribution of QRS abnormalities in the electrocardiographic leads as was pointed out in the preceding discussion of the mechanism of the Q waves of infarction Thus the direction of the unbalanced forces produced by an infarction and perhaps even the time they appear in the QRS interval may well be modified by variations in intraventricular conduction perifocal block and/or coexisting abnormalities of ventricular muscle In view of the uncertainty as to the mechanism of the QRS abnormalities of infarction the meaning of these ab

TABLE 18 – CLASSIFICATION OF ELECTROCARDIOGRAPHIC AND VECTORCARDIOGRAPHIC INFARCTION PATTERNS

	Effective Electrical Location	Direction of Infarction Vector
A Anterior infarctions	Anteroseptal	Left posterior
	Anterior	Posterior
	Anterolateral	Right posterior
	Extensive anterior	Posterior right
B Inferoposterior infarctions	Inferior or diaphragmatic	Superior right
	Posterolateral	Anterior right
	Strictly posterior	Anterior
C Infarctions in combined locations		
D Infarction superimposed on bundle branch block		

normalities in terms of the localization of an infarction must remain in doubt for the time being

For that matter the actual anatomic site of an infarction is largely superfluous information from the standpoint of clinical significance at least insofar as is presently known

The system of nomenclature followed in this text labels an infarction according to its effective electrical position as indicated by the direction of the infarction vector or unbalanced forces created by the infarction (Table 18) As will be recalled in myocardial infarction the unbalanced forces are directed away from the effective electrical site of the infarction For example in anterior myocardial infarction the unbalanced forces created by the infarction which for descriptive convenience can be represented by and referred to as an *infarction vector* are directed posteriorly conversely, in a strictly posterior infarction the infarction vector is directed anteriorly In the preceding examples the effective electrical sites of the two infarctions are in anterior and posterior left ventricular wall respectively although this localization of the infarctions may or may not agree with their anatomic sites as determined at postmortem

Since the ventricular activation process in each type of infarction will be described in terms of the schematic instantaneous VA vectors the following general relationships should be kept in mind

1 When myocardial infarction alters the 0 01 second VA vector there is usually involvement of the apical portion of the interventricular septum
2 When the 0 02 second VA vector is affected there is involvement of apicoanterior or apicodiaphragmatic left ventricular wall (Fig 167)
3 Infarction of anterolateral or diaphragmatic left ventricular free wall generally disturbs both the 0 02 and 0 04 second VA vectors
4 Alterations in the terminal instantaneous vectors such as the 0 06 and 0 08 second VA vectors may well be indicative of infarction of posterior posterobasal or posterolateral left ventricular wall the regions last activated in the left ventricle

Similarly the following general relationships can be considered to exist between the VA vectors and the QRS sÊ loop of the vectorcardiogram

1 The 0 01 second VA vector can be related to the initial deflection of the QRS sÊ loop to the right and/or superiorly
2 The 0 02 second VA vector corresponds roughly

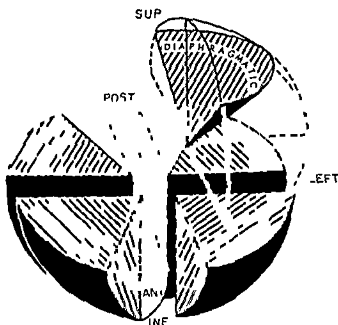


Fig 167 – Approximate range of variation in orientation of the mean 0 02 second instantaneous QRS spatial vector in the normal vectorcardiogram and in vectorcar

to the first half of the efferent limb of the QRS sE loop
3 The 0 04 second VA vecto is analogous to the long axis of maximal instantaneous vector of the QRS sE loop
4 The 0 06 second VA vector can be regarded as representative of the instantaneous vectors form ... f the afferent limb of the ...
VA vector ...

when necessary to represent abnormal terminal QRS forces
... elationships in mind one can ...

The authors have found the one ... 0 02 second instantaneous spatial vector of the QRS sE loop to be of great value in the vector cardiographic diagnosis of myocardial infarction (Fig 107)

TABLE 19 – RELATIONSHIP BETWEEN THE ELECTRICAL SITE OF INFARCTION THE VA VECTORS AFFECTED AND THE RESULTING QRS CHANGES

Electrical Site of Infarction	Direction of Infarction Vector	Instantaneous VA Vectors Affected	Resulting Abnormalities of QRS Complex	Resulting Abnormalities of QRS sE Loop
Anteroseptal	Left posterior	0 01 second septal and 0 02 second apicoanterior VA vectors	QS deflections or abnormal Q waves in leads V V and sometimes V	Leftward and posterior inscription of the initial deflection and early efferent limb of the loop
Anterior	Posterior	0 02 and 0 04-second left ventricular VA vectors	QS deflections or abnormal Q waves in leads V V and V	Posterior displacement of the efferent limb and sometimes the long axis of the loop
Anterolateral	Right, posterior	0 02 and 0 04 second VA vectors	Abnormal Q waves in leads I V and V	Posterior and rightward or medial displacement of the efferent limb and long axis of the loop
Extensive anterior	Right, posterior	0 01 to 0 06-second VA vectors	Abnormal Q waves in leads I and V through V	*Initial inscription of loop to* the right and posterior and rightward or medial displacement of both efferent and afferent limbs and long axis of the loop
Diaphragmatic (inferior)	Superior	0 02 and 0 04 second VA vectors	Abnormal Q waves in leads II III and aVF	Superior displacement of efferent limb and sometimes the long axis of the loop
Posterolateral	Right, anterior	0 02 and 0 04 second VA vectors	Abnormally tall and/or wide R waves in lead V and abnormal Q waves in leads I and V	Rightward and anterior displacement of the early efferent limb and anterior displacement of later efferent limb and long axis of loop
Strictly posterior	Anterior	*0 04- and 0 06-second (or 0 08-second)* VA vectors	Low vibratory RR *deflection or rSR deflection* in leads V and V	*Anterior* displacement of *the long axis and afferent* limb of the *loop*

Anterior Myocardial Infarction

SEVERAL DIFFERENT electrocardiographic and vectorcardiographic infarction patterns are included under the general designation of *anterior myocardial infarction*. In this text the following terms will be employed to label more specifically the various types of anterior infarction: *anteroseptal*, *strictly anterior*, *anterolateral*, and *extensive anterior*. Although the anterior infarctions differ somewhat from one another as to their precise electrical position and consequently their electrocardiographic and vectorcardiographic features, nevertheless they have one characteristic in common—that is, each type of infarction gives rise to unbalanced forces directed to a varying degree posteriorly.

ANTEROSEPTAL INFARCTION

Anteroseptal infarction is usually caused by occlusion of one of the terminal ramifications of the descending branch of the left coronary artery and involves the interapical portion of the interventricular septum and adjacent anterior paraseptal wall of the left ventricle. In the past, two electrocardiographic infarction patterns were ascribed to anteroseptal infarction: one resulting from involvement of both the septum and the paraseptal aspect of the left ventricle and the other from paraseptal involvement only. In this text the authors prefer to designate the first infarction variant "anteroseptal" and the second "strictly anterior," since the vectorcardiographic findings in the two types of anterior infarction are quite distinctive.

The Instantaneous VA Vectors

As will be recalled, the apicoanterior paraseptal portion of left ventricular wall and adjacent septum, which are the usual anatomic sites of involvement in anteroseptal infarction (see also Fig. 168), undergo activation within approximately the first 0.02 second of the QRS interval, so that this type of infarction typically alters the orientation and magnitude of the 0.01 and 0.02 second VA vectors.

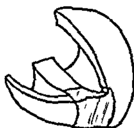
0.01 AND 0.02 SECOND VA VECTORS

Both the 0.01 second septal VA vector and the 0.02 second apicoanterior VA vector are abnormally oriented to the left and posteriorly in anteroseptal myocardial infarction. This has been attributed to an abnormal spread of depolarization through the septum as the result of its involvement by infarction and to the loss of electromotive forces directed anteriorly consequent to apicoanterior wall involvement. Because of the loss of electrical forces directed to the right and anteriorly, the forces directed oppositely become preponderant early in the QRS interval. Thus both 0.01 and 0.02 second VA vectors are displaced posteriorly and to the left. Sometimes it may happen that the loss of anteriorly directed forces produced by activation of apicoanterior left ventricular wall is not sufficient to displace the 0.02 second VA vector into the negative field of lead V_2 even though the vector is located less anteriorly than is normally the case.

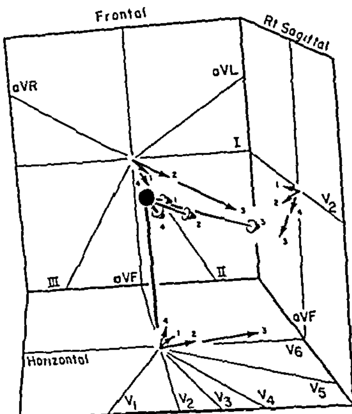
The postinfarction 0.01 second and 0.02 second VA vectors project the following deflections on the precordial leads:

Lead V_1.—Beginning downstroke of a QS deflection.

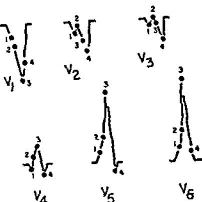
Lead V_2.—Beginning downstroke of a QS deflection (if the 0.02 second VA vector is situated posteriorly just like the 0.01 second VA vector), or a small



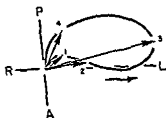
A Anteroseptal Myocardial Infarction

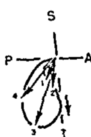


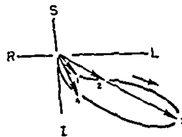
B Instantaneous VA Vectors in Anteroseptal Myocardial Infarction



C QRS Deflections Projected on Scalar Leads







D Planar QRS Loops in Anteroseptal Myocardial Infarction

and to the left The subsequent instantaneous vectors remain unaffected, for all intents and purposes Because of the leftward and posterior deviation of the first two vectors leads V through V record abnormal initial negativity in the form of QS or qrS deflections (C) The scalar limb leads are not depicted in C because as is characteristic in anteroseptal infarction the abnormalities are confined to the precordial leads In D note the absence of the normal initial rightward and anterior deflection of the QRS loop and the anterior concavity in the efferent limbs of the horizontal and sagittal loops

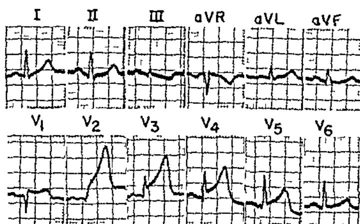


Fig 169 —Electrocardiographic findings in the earliest phase of a developing acute anteroseptal myocardial infarction Although a QS deflection has appeared in lead V_1 and small Q waves in leads V_2 through V_6 the most prominent findings at this stage of the evolution of the infarction pattern are the markedly elevated S–T segments in leads V_1 through V_6 and the very *tall upright T waves in leads V_2 through V* The S–T segment elevation in the anterior precordial leads is compatible with anterior subepicardial injury while the tall upright T waves are probably indicative of subendocardial ischemia If subsequent electrocardiograms had been available leads V_1 through V would probably have shown deepening Q waves and progressive T wave inversion as the S–T segments return to *the isoelectric level*

initial Q wave followed by a minute R wave (if the 0 01 second vector lies posteriorly and the 0 02 second VA vector slightly anteriorly)

Lead V_3 and sometimes lead V_4 —Beginning downstroke of a QS deflection or a small initial Q wave followed by the beginning upstroke of a small or sometimes a larger R wave

Leads V_5 and V_6 —Beginning upstroke of an R wave

0 04 and 0 06 Second VA Vectors

Inasmuch as anterolateral and basal regions of the left ventricle are not affected in anteroseptal myocardial infarction the orientation and magnitude of the electrical forces produced by these regions are normal or at least essentially the same as before infarction so that the precordial leads register the following deflections

Leads V_1 through V_3 or sometimes V_4 —Completion of the S wave of a QS or a qrS complex or of an R wave of a qR deflection

Leads V_5 and V_6 —Completion of the R wave with or without a small terminal S wave

The characteristic deviation of the S–T segments and the T wave abnormalities which accompany the different types of anterior myocardial infarction will be described and the mechanism of these changes reviewed in a separate section at the end of this chapter

QRS Criteria for Diagnosis

The QRS criteria utilized by the authors in making the diagnosis of anteroseptal myocardial infarction are as follows (see also Fig 169)

1 A small abnormal Q wave preceding an rS deflection in V_1 or the presence of a QS deflection in V_1 and leads to the right

TABLE 20 —Orientation of the Mean 0 02 Second and Maximal Mean Instantaneous Vectors of the QRS sÊ Loop in Anteroseptal Infarction

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
Mean 0 02 second instantaneous QRS vector	−15 to −5	−10		+100 to +150	+120		−10 to +30	+5	
Maximal instantaneous QRS vector	−30 to −10	−20		+110° to +170	+135		+10 to +45	+30	

Usual range = range in 85% of cases

2 The presence of QS deflections *or abnormal* Q waves producing qrS qR or qRs deflections in one or more of the next *three leads* (V V₃ and V₄)
3 An abnormal decrease in the relative ampl ude of the R waves *without their disappearance* as the chest electrode is moved to the left of V₁
4 The absence of normal septal Q waves in leads I and V_6

Vectorcardiographic Findings

The average orientation and the extreme ranges of variation in orientation of the mean 0 02 second and maximal mean instantaneous QRS vectors of the hori *zontal sagittal* and frontal loops in anteroseptal in farction are shown in Table 20

Horizontal QRS loop —*In anteroseptal infarc* tion the diagnostic abnormalities if present appear in the horizontal and right sagittal projections of the vectorcardiogram The distinctive feature of the hori zontal QRS loop is the absence of the normal initial deflection of the loop to the right and *anteriorly* In stead with onset of ventricular activation the hori zontal QRS loop moves immediately to the left and *posteriorly* and usually remains in this quadrant dur *ing its* counterclockwise inscription In general the long axis of the horizontal QRS loop (or the maximal instantaneous QRS vector in the horizontal projec *tion*) tends to lie slightly more posterior in antero*septal infarction* than normally Sometimes the effer ent limb of the loop is displaced slightly posteriorly or may show minor posterior bowing *in its early* porti

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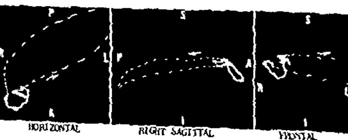
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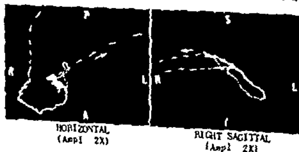
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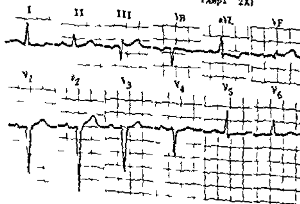


Fig 170 —Electrocardiographic and vectorcardiographic findings in healed anteroseptal *myocardial infarction.*

In the electrocardiogram, the diagnostic a re the QS deflections in leads

of the normal septal Q wave in lead. tice that the S–T segments are isoelectric and the T waves upright in the right precordial the infarction is not

anterior concavity in the first half of the eff t limb of the horizontal QRS loop

partly to the *left and* partly to right—hence the *diphasic* T wave in lead I low upright T wave in lead V *and upright* T waves in the right midprecordial leads

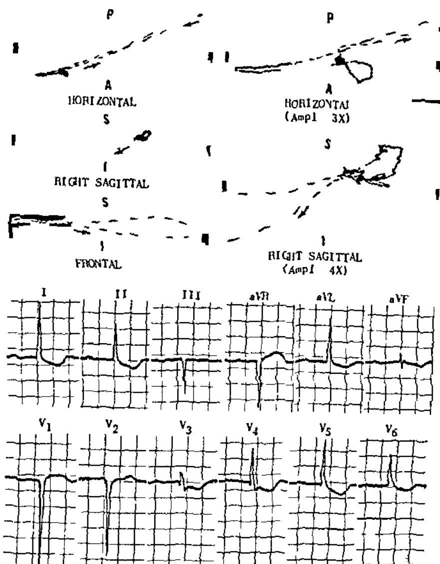


Fig 171 —Electrocardiographic and vectorcardiographic findings in healed anteroseptal myocardial infarction with coexisting left ventricular hypertrophy
In the electrocardiogram the presence of QS deflections in leads V_1 and V_2 and a small Q wave in lead V_3 is indicative of old anteroseptal myocar-

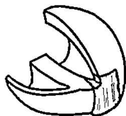
-- existing left ventricular hypertrophy
In the vectorcardiogram the initial deflection of the QRS sÊ loop posteriorly and then to the left is diagnostic of anteroseptal infarction while the elongated and posteriorly and superiorly oriented planar QRS loops and the discordant orientations of the T sÊ and QRS sÊ loops are consistent with left ventricular hypertrophy

a clockwise direction of inscription If the sagittal loop is first inscribed superiorly then it often presents a figure of eight configuration the proximal component of which has a counterclockwise inscription and the larger distal component a clockwise direction of inscription Figure of eight sagittal QRS loops have been a relatively common finding in anteroseptal infarction in our experience

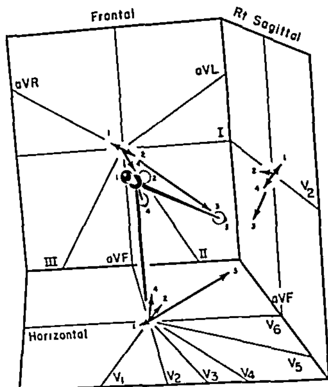
Frontal QRS loop —Since the unbalanced forces created by anteroseptal infarction are directed more or less perpendicular to the frontal plane the frontal QRS loop does not show diagnostic changes Although the frontal QRS loop is inscribed initially to the left this finding has little diagnostic value because the normal frontal QRS loop often appears to proceed directly to the left (Figs 170 and 171)

STRICTLY ANTERIOR INFARCTION

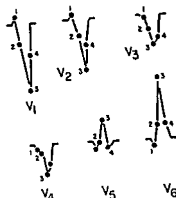
The unbalanced forces created by anterior myocardial infarction are oriented almost directly posteriorly as if the site of the infarction were in paraseptal anterior left ventricular wall which more often than not proves to be the case on pathologic study (However as previously pointed out the electrical and anatomic sites of an infarction need not invariably be identical) The anterior wall of the left ventricle is activated relatively early in the QRS interval Consequently the failure of the infarcted anterior wall to generate potentials during the time it normally is undergoing activation permits oppositely directed electrical forces to become preponderant For this reason the electrical effects of an anterior infarction



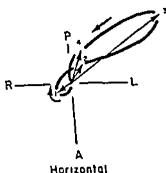
Ⓐ Anterior Myocardial Infarction

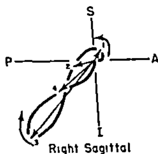


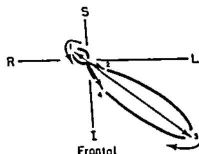
Ⓑ Instantaneous VA Vectors in Anterior Myocardial Infarction



Ⓒ QRS Deflections Projected on Scalar Leads







Ⓓ Planar QRS Loops in Anterior Myocardial Infarction

posteriorly In fact, the long axis of the horizontal QRS loop is deviated farther posteriorly than normally Note also that the direction of inscription of the sagittal QRS loop is counterclockwise for the initial part of the QRS interval

are evidenced in the early part of the QRS deflection or QRS sE loop

The Instantaneous VA Vectors

The electrical forces produced by septal and ventricular activation in strictly anterior myocardial infarction can be presented in simplified manner in terms of the instantaneous vectors (see also Fig 172)

0 01 Second VA Vector

Infarction restricted to the anterior aspect of the left ventricle does not disturb the normal orientation of the 0 01 second VA vector to the right and anterior in contrast with the situation in anteroseptal infarction Thus the appearance of the 0 01 second VA vector in strictly anterior myocardial infarction is accompanied in the precordial leads by inscription of the following deflections

Lead V_1 —Upstroke of a small initial R wave

Lead V_2 —Beginning upstroke of a small initial R wave

Leads V_3 and V_4 —Depending on the anterior extent of the 0 01 second VA vector these leads may record a small initial R wave a Q wave or an isoelectric segment

Leads V_5 and V_6 —Downstroke of a small initial Q wave

0 02 and 0 04 Second VA Vectors

Normally these vectors are determined largely by activation of left ventricular free wall In anterior infarction apicoanterior wall and adjacent anterior wall of the left ventricle are rendered electrically inert with the result that opposing forces oriented posteriorly dominate the electrical field of the heart from 0 02 to 0 04 second or longer after onset of ventricular activation The postinfarction 0 02 and 0 04 second VA vectors which are the resultants of the corresponding preinfarction VA vectors and the posteriorly directed infarction vector are both displaced farther posteriorly The persisting leftward deviation of the 0 04 second VA vector probably reflects activation of uninvolved anterolateral and lateral walls of the left ventricle The postinfarction 0 02 and 0 04 second VA vectors produce the following deflections in the precordial leads

Leads V_1 and V_2 —Downstroke of a deep S wave Inasmuch as the normal 0 02 second VA vector plays a more important role in the genesis of the initial R wave in lead V_2 than in lead V_1 the posterior displacement of this vector in anterior infarction frequently causes the R wave in lead V_2 to have a lower amplitude relative to the total QRS amplitude than that of the R wave in lead V_1

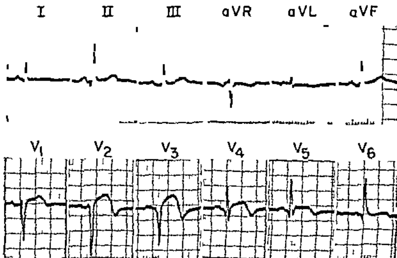


Fig 173 —Electrocardiographic findings in acute anterior myocardial infarction The salient features are a normal initial R wave in lead V_1 and a relatively smaller R wave in V_2 a QS deflection in lead V_3 a promi

of myocardial necrosis the S-T segment elevation of anterior subepicardial injury and the T wave changes of transmural ischemia—all of which add up to acute anterior infarction

Leads V_3 and V_4 —Downstroke of a deep S wave following a minute initial R wave or the downstroke of a Q wave of 0 04 second duration When these leads register a small initial R wave its relative amplitude is less than that in leads V_2 and/or V_1 Occasionally particularly in old anterior infarctions leads V_1 through V_4 may all display rS deflections In this event the relative amplitude of the initial R wave decreases from right to left in two or more of the leads in contrast with the normal progressive increase in the R/S ratio from right to left across the precordium Sometimes the only evidence of an old infarction may be the presence of a small initial Q wave preceding a small R wave and a deep S wave (qrS deflection) In general a Q wave (even if relatively small and narrow) which precedes the r wave of an rS deflection recorded in a right or midprecordial lead should be

viewed with suspicion, particularly if the Q wave is r n n om nent than the Q waves in lead V_5 or V_6 or

hypertrophy

Leads V_5 and V_6 —Completion of the Q wave and upstroke of the following R wave Sometimes particularly in lead V_5 the Q wave may be suspiciously deep and/or wide and the R wave *of somewhat low* amplitude but typically in strictly anterior infarction leads V_5 and V_6 do not show diagnostic changes involving the QRS deflections

0 06-Second VA Vectors

In the vast majority of anterior infarctions the terminal QRS forces are not characteristically disturbed However occasionally the 0 06-second VA vector or subsequent instantaneous vectors may be directed to the right, away from the 0 04 second or maximal instantaneous vector Grant has postulated that in some cases peri infarction block may be the mechanism responsible for this variation (Peri infarction block is discussed in Chapter 21)

Leads V_1 and V_2 —Completion of the S wave of an rS deflection

Leads V_3 and V_4 —Completion of the S wave of a QS or Qrs deflection or completion of the R wave of a Qr or rSR deflection

Leads V_5 and V_6 —Completion of the R wave of a qR deflection or the small S wave of a qRs deflection

QRS Criteria for Diagnosis

1 The presence of initial R waves in leads V_1 or V_{3R} and of abnormal Q waves in one or more precordial leads to the left of V_1 (i e leads V V_3 or V_4)

qrS deflections or abnormally wide and/or deep Q waves of QR or QRs deflections

2. A right to-left decrease in the relative amplitudes of the R waves without their disappearance in precordial leads to the left of lead V_1

3 The presence of normal septal Q waves in leads I V_5 and V_6 (As will be recalled the absence of normal Q waves in the above leads is a common observation in anteroseptal infarction The absence of abnormally wide and/or deep Q waves in leads I V_5 and V_6 excludes the possibility of anterolateral as opposed to strictly anterior infarction [Fig. 173])

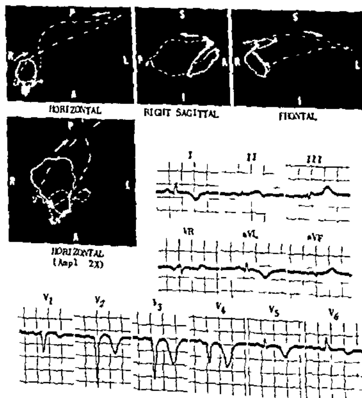


Fig 174 —Electrocardiographic and vectorcardiographic findings in recent anterior myocardial infarction

In the electrocardiogram lead V shows an initial R wave and lead V a small septal wave but intervening leads V through V display QS deflections isoelectric S–T segments and deeply and symmetrically inverted T waves This record represents a slightly later stage in the evolution of an anterior infarction than that in Figure 173

In the vectorcardiogram there is striking posterior displacement of the efferent limb of the QRS sÊ loop and a reversed direction of inscription of the sagittal QRS loop Note the large round and posteriorly directed T loop in the horizontal projection This is a typical finding in vectorcardiograms of patients with deeply and symmetrically inverted T waves of transmural ischemia.

are evidenced in the early part of the QRS deflection or QRS sÊ loop

The Instantaneous VA Vectors

The electrical forces produced by septal and ventricular activation in strictly anterior myocardial infarction can be presented in simplified manner in terms of the instantaneous vectors (see also Fig 172)

0 01 Second VA Vector

Infarction restricted to the anterior aspect of the left ventricle does not disturb the normal orientation of the 0 01 second VA vector to the right and anterior in contrast with the situation in anteroseptal infarction Thus the appearance of the 0 01 second VA vector in strictly anterior myocardial infarction is accompanied in the precordial leads by inscription of the following deflections

Lead V_1 —Upstroke of a small initial R wave

Lead V_2 —Beginning upstroke of a small initial R wave

Leads V_3 and V_4 —Depending on the anterior extent of the 0 01 second VA vector these leads may record a small initial R wave a Q wave or an isoelectric segment

Leads V_5 and V_6 —Downstroke of a small initial Q wave

0 02 and 0 04 Second VA Vectors

Normally these vectors are determined largely by activation of left ventricular free wall In anterior infarction apicoanterior wall and adjacent anterior wall of the left ventricle are rendered electrically inert with the result that opposing forces oriented posteriorly dominate the electrical field of the heart from 0 02 to 0 04 second or longer after onset of ventricular activation The postinfarction 0 02 and 0 04 second VA vectors which are the resultants of the corresponding preinfarction VA vectors and the posteriorly directed infarction vector are both displaced farther posteriorly The persisting leftward deviation of the 0 04 second VA vector probably reflects activation of uninvolved anterolateral and lateral walls of the left ventricle The postinfarction 0 02 and 0 04 second VA vectors produce the following deflections in the precordial leads

Leads V_1 and V_2 —Downstroke of a deep S wave Inasmuch as the normal 0 02 second VA vector plays a more important role in the genesis of the initial R wave in lead V_2 than in lead V_1 the posterior displacement of this vector in anterior infarction frequently causes the R wave in lead V_2 to have a lower amplitude relative to the total QRS amplitude than that of the R wave in lead V_1

Leads V_3 and V_4 —Downstroke of a deep S wave following a minute initial R wave or the downstroke of a Q wave of 0 04 second duration When these leads register a small initial R wave its relative amplitude is less than that in leads V_2 and/or V_1 Occasionally particularly in old anterior infarctions leads V_1 through V_4 may all display rS deflections In this event the relative amplitude of the initial R wave decreases from right to left in two or more of the leads in contrast with the normal progressive increase in the R/S ratio from right to left across the precordium Sometimes the only evidence of an old infarction may be the presence of a small initial Q wave preceding a small R wave and a deep S wave (qrS deflection) In general a Q wave (even if relatively small and narrow) which precedes the r wave of an rS deflection recorded in a right or midprecordial lead should be

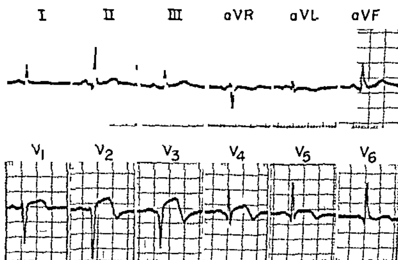


Fig 173 —Electrocardiographic findings in acute anterior myocardial infarction The salient features are a normal initial R wave in lead V_1 and a relatively smaller R wave in V_2 a QS deflection in lead V_3 a prominent Q wave in lead V_4 elevated S-T segments in leads V_1 through V_6 and inverted T waves in leads V_3 through V_6 Thus the midprecordial leads show the QRS changes of myocardial necrosis the S-T segment elevation of anterior subepicardial injury and the T wave changes of transmural ischemia—all of which add up to acute anterior infarction

The average orientation and the extreme ranges of variation of orientation of the mean 0 02 second and maximal mean instantaneous *QRS vectors of the hori*zontal right sagittal, and frontal loops in anterior infarction are shown in *Table 21*

HORIZONTAL QRS LOOP —In anterior infarction the horizontal QRS loop usually conforms in appearance to one of the *following pattern variations*

1 The least prominent abnormality of the horizontal QRS loop may consist solely of an anterior concavity *or posterior bowing of the efferent limb* The inscription of the loop is otherwise not too different from the normal The long axis of the loop is situated *posteriorly but not always to an abnormal degree* On the other hand the posterior displacement of the 0 02 second instantaneous vector is a finding relatively rarely observed in normal vectorcardiograms

2 Frequently in anterior infarction the horizontal QRS loop is initially written in a clockwise direction to the right and anteriorly or sometimes posteriorly and then the remainder of the loop is inscribed in a counterclockwise direction posteriorly and to the left. The efferent limb is displaced posteriorly and/or shows an anterior concavity Both the long axis of the loop and the 0 02 second instantaneous vector tend to be oriented more posteriorly than in the pattern previously described

3 Another variant loop pattern observed in anterior infarction is characterized by counterclockwise inscription of the initial deflection of the horizontal *QRS loop to the right* and anteriorly and then clockwise inscription of the remainder of the loop to the left and far posteriorly Thus the horizontal QRS

eight" On the average the *maximal mean instanta*neous vector and mean 0 02 second instantaneous vector of the horizontal QRS loop occupy a more *posterior position in this pattern variant than in any* of the others observed in strictly anterior infarction

4 Finally the horizontal QRS loop is sometimes written in a clockwise *direction initially to the right* and anteriorly or posteriorly but in contrast with the QRS loop configuration described above in paragraph 2 the rest of the loop continues to be inscribed in a



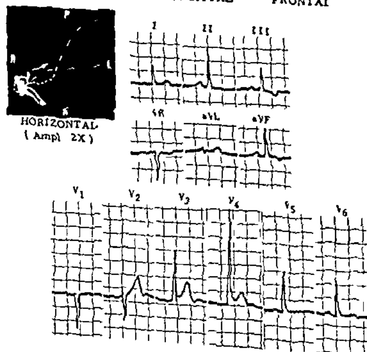


Fig 176 —Well localized anterior myocardial infarction which can be diagnosed *only in the vec*torcardiogram The electrocardiogram shows no definite evidence of anterior myocardial infarction The significance of the elevated S–T segments in leads V and V with *reference to the diagnosis of infarc*tion would be impossible to deter*mine* without serial tracings However the marked anterior concavity in the efferent limb of the QRS sE loop of the vectorcardiogram is indicative of a well localized anterior infarction

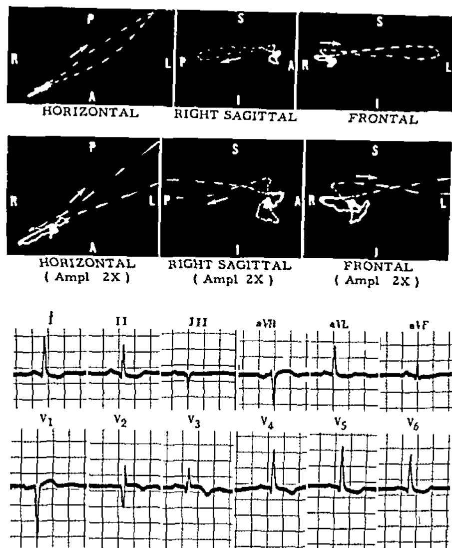


Fig 175 —Electrocardiographic and vectorcardiographic findings in healed or old anterior myocardial infarction The QRS abnormalities diagnostic of infarction are confined chiefly to leads V_3 and V_4 of the electrocardiogram The horizontal QRS loop of the vectorcardiogram is strikingly abnormal in that its direction of inscription is completely reversed Clockwise inscribed horizontal QRS loops with *normal rightward and anterior* initial deflections are frequently observed in anterior myocardial infarction There is an S–T vector which is best demonstrated *in the sagittal* and frontal projections It is directed to the right and superiorly and therefore projects depressed S–T segments on leads I aVL and left precordial leads The T sÊ loop is oriented almost 180 away from the QRS sÊ loop

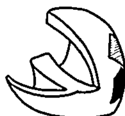
Vectorcardiographic Findings

Although the QRS sÊ loop in strictly anterior infarction tends to be variable in appearance as will be described below the loop in most instances will display at least the following two salient features (1) In contrast with the horizontal QRS loop in anteroseptal infarction the corresponding loop in anterior infarction is written initially to the right and usually anteriorly or occasionally slightly posteriorly (2) The efferent limb of the QRS sÊ loop either in whole or in part is written abnormally far posteriorly

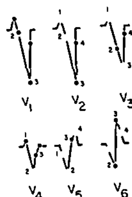
TABLE 21 —ORIENTATION OF THE MEAN 0 02 SECOND AND MAXIMAL MEAN INSTANTANEOUS VECTORS OF THE QRS sÊ LOOP IN STRICTLY ANTERIOR INFARCTION

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
Mean 0 02 second instantaneous QRS vector	−100 to −5	−30	−60 to −10	+100 to −110	+170	+110 to −150	−120 to +85	+15	−70 to +65
Maximal mean instantaneous QRS vector	−60 to −10	−30	−35 to −10	+110 to −170	+155	+130 to 180	−5 to +45	+15	+5 to +35

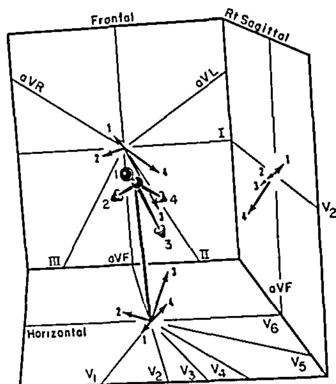
Usual range = range in 85% of cases



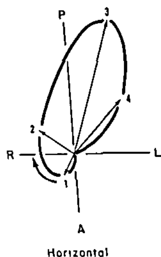
A Anterolateral Myocardial Infarction

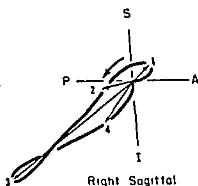


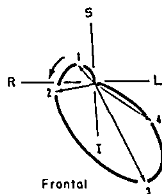
C QRS Deflections Projected on Scalar Leads



B Instantaneous VA Vectors in Anterolateral Myocardial Infarction







D Planar QRS Loops in Anterolateral Myocardial Infarction

Fig 177 –(*Legend on facing page*)

clockwise direction to the left and posteriorly Thus the efferent limb of the horizontal loop is shifted posterior to the afferent limb Both maximal mean and mean 0 02 second instantaneous vectors lie abnormally far posteriorly

RIGHT SAGITTAL QRS LOOP —In anterior infarction the sagittal QRS loop confirms the marked posterior displacement of the long axis and mean 0 02 second instantaneous vector of the QRS sÊ loop In about 75% of the cases the sagittal QRS loop is inscribed either entirely or in part in a counterclockwise direction In the latter event the loop has a figure of eight configuration the proximal component being counterclockwisely inscribed and the distal component clockwisely inscribed A figure-of eight sagittal QRS loop is probably the most common configuration observed in anterior infarction

FRONTAL QRS LOOP —The frontal projection of the QRS sÊ loop exhibits no characteristic abnormalities in localized anterior infarction (Figs 174–176)

ANTEROLATERAL INFARCTION

By and large infarctions of the lateral wall of the left ventricle can be placed in either of two categories anterolateral infarction or posterolateral infarction In *anterolateral infarction* the infarction vector (representing the unbalanced forces produced by the infarction) is directed somewhat posteriorly but for the most part to the right Thus abnormal Q waves are projected typically on leads I aVL V_5 and V_6 and sometimes on lead V_4 In *posterolateral infarction* the infarction vector is directed somewhat anteriorly but *mainly to the right In the electrocardiogram* leads V_6 and V_7 typically register abnormal Q waves and leads V_{3R} V_1 and V_2 record relatively tall R waves of 0 04 second or more duration In our experience virtually all lateral infarctions fall into one of these two groups and it does not seem necessary to distinguish a third category of *strictly lateral infarction* Posterolateral infarction is discussed in Chapter 20 dealing with inferoposterior myocardial infarctions

The Instantaneous VA Vectors

The electrical forces produced during ventricular activation in anterolateral infarction can be presented in simplified manner in terms of the instantaneous VA vectors (see also Fig 177)

0 01 SECOND VA VECTOR

Inasmuch as septal depolarization is ordinarily not affected in anterolateral infarction the 0 01 second VA vector is of the same order of magnitude and the same direction as normal The 0 01 second VA vector is therefore directed to the right anteriorly and either superiorly or inferiorly and projects the following deflections on leads V_1 V_5 V_6 I and aVL

Lead V_1 —Upstroke of an initial R wave

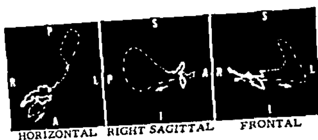
Leads V_5 V_6 I and aVL —Beginning downstroke of a Q wave

0 02 AND 0 04 SECOND VA VECTORS

The anterolateral wall of the left ventricle which undergoes activation between 0 02 and 0 04 second of the QRS interval is electrically inert and therefore no longer produces potentials to counterbalance oppositely directed electrical forces The forces left unbalanced can be represented by an infarction vector which is directed to the right and posteriorly The postinfarction 0 02 second VA vector which is the resultant of the infarction vector and the corresponding preinfarction 0 02 second vector (normally oriented to the left and anteriorly) is therefore displaced to the right and anteriorly or slightly posteriorly Similarly the postinfarction 0 04 second VA vector which is the resultant of the infarction vector and the preinfarction 0 04 second vector situated to the left and posteriorly assumes a more medial and posterior position in anterolateral infarction

Lead V_1 —Completion of an initial R wave of increased amplitude and duration (if the 0 02 second VA vector lies to the right and anteriorly) or as is more commonly the case the beginning downstroke

Fig 177 —Instantaneous VA vectors in the anterolateral myocardial infarction farction is indicated in **A** As shown in **B** instantaneous VA vectors 2 and 3 are di poster there waves ap malities ent limb



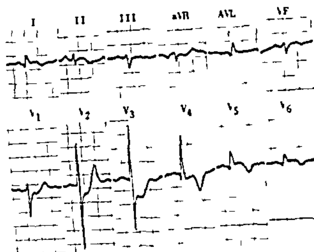
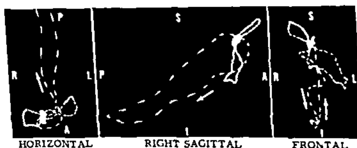


Fig 179 – Electrocardiographic and vectorcardiographic findings in acute anterolateral myocardial infarction

Because the electrocardiogram was recorded quite early in the evolution of the infarction pattern the Q wa es in leads I aVL, and V ... V ... ll relatively small however ...

se ... ri ... tl ...

vector in the horizontal projection ... abc ... (normal = rarely to the right of +1 ... en ... tic ... a ... large S–T vector directed to the left and posteriorly and the rightward and anterior orientation of the T sÊ loop



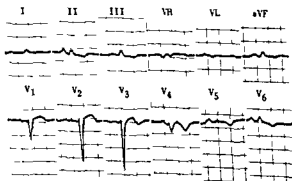


Fig 180 – Electrocardiographic and vectorcardiographic findings in anterolateral infarction of uncertain duration Although diagnostic Q waves do not appear in the electrocardiogram the ...

diogram the QRS sÊ loop is oriented directly posteriorly and inferiorly The efferent limb of the horizontal QRS loop lies to the right of the afferent limb while in the frontal QRS loop the efferent limb is inscribed irregularly *crossing the afferent limb at three* points It must be pointed out that the vectorcardiogram was recorded at maximal amplification and so the planar QRS loops actually enclose extremely mall areas compared with normal loops The medial or rightward displacement of the efferent limb of the QRS sÊ loop and the greatly diminished magnitude of the instantaneous vectors of the QRS sÊ loop are diagnostic of anterolateral infarction The T sÊ loop is oriented to the right superiorly and anteriorly and there is no discernible S–T vector Consequently the age of the infarction cannot be determined from the vectorcardiogram Note that the P sÊ loop in the frontal projection is almost as large as the frontal QRS loop

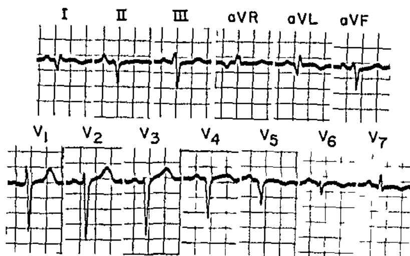


Fig 178 —Electrocardiographic findings in anterolateral myocardial infarction of uncertain duration The diagnostic features are the wide deep Q waves in leads I and aVL the QS deflections in leads V and V_6 the small although significant Q wave preceding the embryonic R wave in lead V the relatively tall wide R wave in lead V_1 with progressive diminution of relative R wave amplitude from right to left until the R wave disappears entirely the slight coving of the S–T segments and the inverted T waves in leads I aVL and V_6 through V_7 In the absence of definite S–T segment ... the infarction

of an S wave (if the 0 02 second vector is located to the right and posteriorly)

Leads I aVL V_5 and V_6 —Completion of abnormally deep and wide Q waves

0 06 Second VA Vector

Since posterobasal areas of the left ventricle are usually spared in anterolateral infarction the 0 06 second and subsequent instantaneous vectors generally occupy much the same position as they do normally although occasionally they may be displaced farther posteriorly and sometimes to the right of the midline

Lead V_1 —Completion of a terminal S wave

Leads I aVL V_5 and V_6 —A terminal R wave of reduced amplitude (if the terminal vectors are situated to the left and posteriorly [Fig 178] or completion of a QS deflection (if all VA vectors after 0 01 second lie to the right of the midline)

Lead aVL in Diagnosis

Although occasionally the only diagnostic evidence of a high anterolateral infarction may consist of wide deep Q waves in lead aVL Q waves of equal prominence are frequently recorded in lead aVL in normal electrocardiograms Proponents of the unipolar concept of electrocardiography attribute this finding in normal subjects to backward rotation of the cardiac apex and vertical heart position which results in lead aVL recording initial negative potentials from the left ventricular cavity and later positive potentials from the epicardial surface of basal left ventricular wall Since the right shoulder lead electrode also faces the back of the heart during atrial activation and ventricular repolarization lead aVL also displays inverted P and T waves Consequently the criteria recommended by Goldberger for Q wave abnormality in lead aVL stipulate that P and T waves must be upright before a prominent Q wave in this lead can be considered abnormal In general the experience of the authors has been that in the diagnosis of infarction the findings in lead aVL are often confusing By and large lead I and/or lead V_6 record the transverse (X) component of the cardiac vector more faithfully than does lead aVL The reason for this is inherent in the direction of the lead axis of lead aVL The axis of lead aVL is situated along the −30° to +150 axis of the frontal reference frame and obviously is neither transverse nor vertical but rather is a hybrid responding disproportionately to both X and Y (vertical) components of the cardiac vector Consequently lead aVL records Q waves when the initial and early instantaneous QRS vectors lie between +60 and +90 even though these vectors are situated to the left of the midline and project no Q waves on lead I On the other hand lead aVL fails to register Q waves when the initial and early instantaneous vectors are situated to the right between −90 and −120 even though these vectors project Q waves on lead I Thus lead aVL fails completely to discriminate between vectors situated to the right or left when these vectors lie in either the +60 to +90 or the −90 to −120 segment of the frontal reference frame Moreover initial vectors oriented to the right and inferiorly (a common occurrence in normal persons) project more prominent Q waves on lead aVL than on lead I In fact it is difficult to differentiate such Q waves from those occurring in lead aVL in anterolateral infarction as witnessed by the number of criteria which a Q wave in this lead must

TABLE 22 —ORIENTATION OF THE MEAN 0 02-SECOND AND MAXIMAL MEAN INSTANTANEOUS VECTORS OF THE QRS sÊ LOOP IN ANTEROLATERAL INFARCTION

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	A	Usual Range	Extreme Range	A	Usual Range	Extreme Range	A	Usual Range
Mean 0 02 second instantaneous QRS vector	+95 to −60	−160	+95 to −110	+130 to +10	−100	−165 to +10	+90 to −90	−155	+90 to −120
Maximal mean instantaneous QRS vector	−90 to −50	−70	−90 to −65	+150 to −170	+170	+170 to −170	−60 to +95	+10	+5 to +95

Usual range = range in 85% of cases.

the midline In anterolateral infarction the long axis of the loop or the maximal instantaneous QRS vector in the horizontal projection ordinarily is situated between −90 and −50 in the horizontal reference frame

3 The mean 0 02 second instantaneous QRS vector in this projection usually *lies well to the right* and either anteriorly or posteriorly in contrast with its normal orientation to the left and anteriorly Occasionally in anterolateral infarction this vector is located to the left and far posteriorly

4 The horizontal QRS loop may have a clockwise or counterclockwise direction of inscription depending apparently on the degree of posteromedial displacement of the efferent limb Frequently QRS loops inscribed in a counterclockwise direction present a figure-of eight configuration

5 Occasionally in our series of anterolateral infarctions moderate conduction slowing was observed in the afferent limb of the QRS sÊ loop in all three projections although this was seldom of such a degree as to produce QRS prolongation to 0 12 second

RIGHT SAGITTAL QRS LOOP —The QRS sÊ loop in this projection confirms the marked posterior orientation already described in the horizontal projection The direction of inscription may be either clockwise or counterclockwise

FRONTAL QRS LOOP —Unlike anteroseptal and anterior infarctions which cause *no recognizable abnormalities* of the frontal QRS loop anterolateral infarction typically produces one or both of the following abnormalities in the frontal projection

1 The initial and early portion of the loop is written farther to the right than normal

2 The remainder of the loop is usually inscribed in a counterclockwise direction to the left and often somewhat *superiorly* More often than not the efferent limb of the loop after moving for a time horizontally and to the left then turns abruptly superiorly and medially This sudden change in the course of the loop possibly reflects involvement of portions of the lateral ventricular wall activated relatively late in the QRS interval (Figs 179–181)

EXTENSIVE ANTERIOR INFARCTION

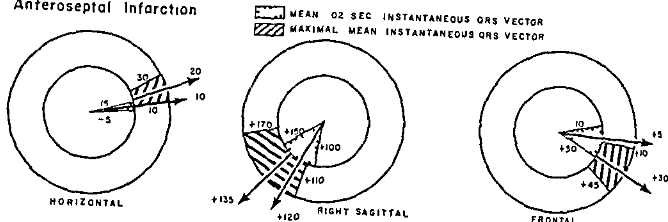
Extensive anterior infarction usually corresponds to occlusion of the anterior descending branch of the left coronary artery

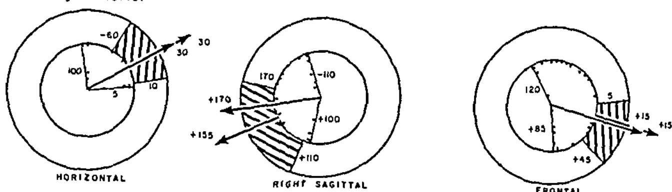
The Instantaneous VA Vectors

Extensive anterior myocardial infarction is essentially a combination of anteroseptal, anterior and anterolateral infarctions and is therefore accompanied by displacement of the 0 01 0 02 and 0 04 second VA vectors to the right and posteriorly (Fig 182) The 0 06 second VA vector and subsequent instantaneous vectors are situated posteriorly and either slightly to the right or left of the midline Thus leads I aVL and V_1 through V_6 record abnormal Q waves

Vectorcardiographic Findings

The QRS sÊ loop in extensive anterior infarction typically shows the entire gamut of abnormalities de





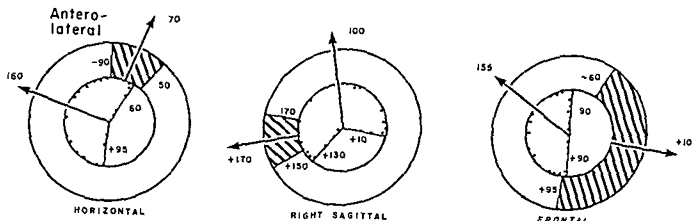


Fig 181 —Range of variation and average orientation of the mean 0 02 second and the maximal mean instantaneous QRS vectors of the vectorcardiographic QRS sE loop in the various types of anterior myocardial infarction

satisfy to be considered abnormal (see Criteria of Q Wave Abnormality in Chapter 18)

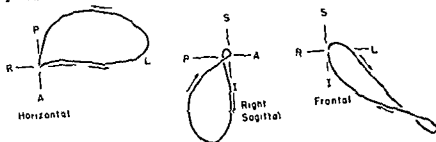
Vectorcardiographic Findings

The average orientation and the extreme ranges of variation in orientation of the mean 0 02 second and maximal mean instantaneous QRS vectors of the horizontal right sagittal and frontal loops in anterolateral infarction are shown in Table 22

Horizontal QRS loop —The salient diagnostic abnormalities presented by the QRS sE loop in the horizontal projection are as follows

1 The initial and early portions of the horizontal QRS loop extend farther to the right and anteriorly than normally is the case and this initial deflection of the loop usually encloses a relatively large area
2 The body of the horizontal QRS loop is written far posteriorly and slightly to the left or right of

I Anteroseptal Infarction



II Strictly Anterior Infarction

A

P R L A Horizontal — S P A I Right Sagittal — S R L I Frontal

B

P R L A Horizontal — S P A I Right Sagittal — S R L I Frontal

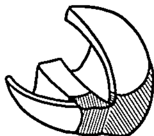
C

P R L A Horizontal — S P A I Right Sagittal — S R L I Frontal

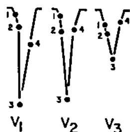
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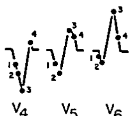
P R L A Horizontal — S P A I Right Sagittal — S R L I Frontal

Fig 183.—Planar QRS loop patterns observed vectorcardiographically in the various types of anterior myocardial infarction (I) anteroseptal infarction (II) strictly anterior infarction and its pattern variants (A–D) (*continued*)

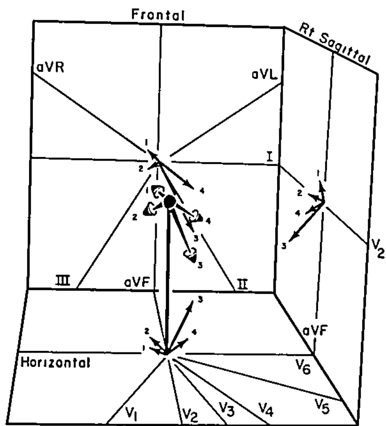


A Extensive Anterior Myocardial Infarction



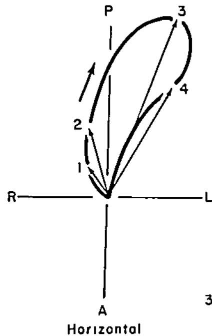


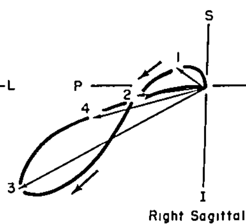
C QRS Deflections Projected on Scalar Leads

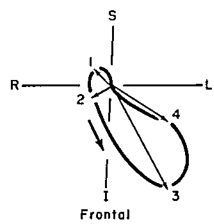


B Instantaneous VA Vectors in Extensive Anterior Myocardial Infarction

D Planar QRS Loops in Extensive Anterior Myocardial Infarction





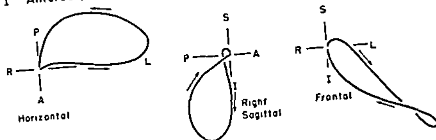


in

ar

and to the right ... can be seen to present a combination of the features of anteroseptal and anterolateral infarction

D can be seen to present a combination of the features of anteroseptal and anterolateral infarction



II Strictly Anterior Infarction

A
P R L A Horizontal
S P A I Right Sagittal
S R L I Frontal

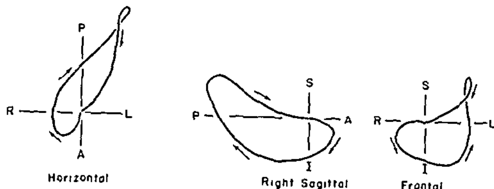
B
P R L A Horizontal
S P A I Right Sagittal
S R L I Frontal

C
P R L A Horizontal
S P A I Right Sagittal
S R L I Frontal

D
P R L A Horizontal
S P A I Right Sagittal
S R L I Frontal

Fig 183 —Planar QRS loop patterns observed vectorcardiographically in the various types of anterior myocardial infarction (I) anteroseptal infarction (II) strictly anterior infarction and its pattern variants (A–D) (*continued*)

III Anterolateral Infarction



IV Extensive Anterior Infarction

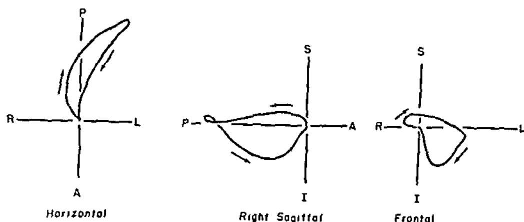


Fig 183 *(cont)*—(III) anterolateral infarction and (IV) extensive anterior infarction

scribed for anteroseptal strictly anterior and anterolateral infarctions (Fig 183) Generally the initial deflection and efferent limb of the QRS sÊ loop are written to the right posteriorly and somewhat superiorly and the loop then returns on the left posteriorly and inferiorly *Thus abnormal Q waves are projected* on all six precordial leads and leads I and aVL

The average orientation and the extreme ranges of variation in orientation of the mean 0 02 second and maximal mean instantaneous QRS vectors of the horizontal sagittal and frontal loops in extensive anterior *infarction are shown in Table 23*

TABLE 23 —Orientation of the Mean 0 02 Second and Maximal Mean Instantaneous Vectors of the QRS sÊ Loop in Extensive Anterior Infarction

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
Mean 0 02 second instantaneous QRS vector	−50 to −30	−40		+110 to +130	+120		+50 to +70	+60	
Maximal mean instantaneous QRS vector	−60 to −45	−50*		−170 to −160	−165		−50 to +10	−20	

Usual range = range in 85% of cases

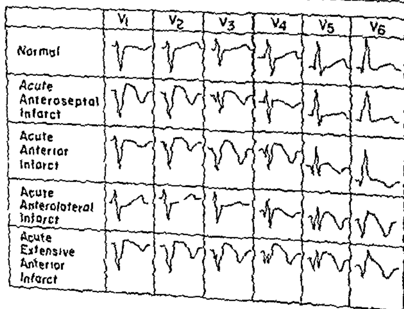
S–T VECTOR AND VENTRICULAR REPOLARIZATION IN ACUTE PHASE OF ANTERIOR INFARCTION

injury Thus as will be recalled at [illegible] ventricular activation *the subepicardial layer of ventricular muscle* anteriorly retains some of its dipoles because the depolarization wave has been partially or wholly *blocked at the* boundaries of the injured region The latter therefore *comes to be positively* charged with respect to deeper layers of muscle and a *systolic current of injury* flows between the two The electrical field established by the current of injury *can be represented by an injury or* S–T vector which *in anterior myocardial infarction* is directed toward the effective electrical site of *epicardial* injury —that is anteriorly Consequently the S–T vector projects positive voltages *in the form of* elevated S–T *segments on one or more* of the anterior precordial *leads* In the *vectorcardiogram the presence of* an S–T vector in anterior infarction is evidenced by failure of the QRS sE loop to return to its point of origin *in the horizontal and right sagittal projections* the terminus of the QRS sE *loop being displaced* anteriorly The more exact *orientation of the* S–T vector *in each of the four types of anterior infarction* is *indicated* in Table 24

As the intense current of injury subsides during the course of an anterior infarction the effects of transmural ischemia begin to appear with increasing prominence *in the electrocardiogram* and vectorcardiogram As indicated previously *transmural ischemia* apparently delays recovery in subepicardial muscle to a *greater degree than in subendocardial* muscle

TABLE 24 —RELATIONSHIP BETWEEN THE EFFECTIVE ELECTRICAL SITE OF SUBEPICARDIAL INJURY AND TRANSMURAL ISCHEMIA AND THE DIRECTION OF THE S–T VECTOR AND THE T sE LOOP IN ANTERIOR MYOCARDIAL INFARCTION

Effective Electrical Site of Injury and Ischemia	Direction of S–T Vector	Orientation of T sE Loop
Anteroseptal	Right, anterior	Left, posterior
Anterior	Anterior and slightly to the left	Posterior and *slightly to the right*
Anterolateral	Left anterior	Right, posterior
Extensive anterior	Anterior or left and anterior	Posterior right



thereby reversing the direction in which repolarization spreads through the ischemic ventricular wall When the anterior wall of the left ventricle is the site of transmural ischemia as is the case in an anterior infarction repolarization forces produced in this region are directed posteriorly (just the opposite of normal) and continue to be generated for a time after repolarization *elsewhere in the ventricles has been com*pleted The instantaneous T vectors which are the resultant of these abnormal forces arising in the anterior ventricular wall and of normal forces produced elsewhere tend to be displaced posteriorly away from the effective site of transmural ischemia The specific orientation of the T sÊ loop in each of the four types of anterior infarction is presented in Table 24 but in general during the ischemic phase of an anterior infarction the instantaneous T vectors project symmetrical and deeply inverted T waves on one or more anterior chest leads and cause the T sÊ loop of the vectorcardiogram to rotate posteriorly

Since the evolution of S–T segment and T wave changes in myocardial infarction has already been described in Chapter 18, it will not be described specifically for each type of infarction Let it suffice *to say that early in an anterior infarction the S–T* segments are for a time elevated in one or more precordial leads and that as the S–T segments subsequently return to the isoelectric base line the T waves in the same leads or lead become increasingly more deeply inverted In the healed stage of an anterior infarction the S–T segments are isoelectric but the T waves may be of normal appearance low upright or asymmetrically inverted

For a resume of the electrocardiographic findings in the four types of anterior infarction see Table 24

Inferoposterior Myocardial Infarction

An inferoposterior infarction is usually due to an occlusion of the right coronary artery or circumflex artery and may have any of the following effective electrical locations (1) inferior or diaphragmatic wall of the left ventricle (2) posterolateral wall of the left ventricle or (3) strictly posterior (posterobasal or infra atrial) wall of the left ventricle. Like the anterior infarctions inferoposterior infarctions are distinguished one from another by their electrical location, as evidenced by the direction of the unbalanced forces they produce

DIAPHRAGMATIC (INFERIOR) INFARCTION

Infarction of the diaphragmatic or inferior wall of the left ventricle abolishes the leftward and inferiorly directed QRS forces normally produced by this region with the result that forces acting in the opposite direction become electrically preponderant In effect, it is as if new forces of equal magnitude but opposite direction were added to the balance of cardiac forces These new forces can be visualized as an infarction vector which in diaphragmatic infarction exerts its effect on the electrical field of the heart primarily in a superior direction—that is along a vertical axis perpendicular to the horizontal plane The abnormalities produced by diaphragmatic infarction are therefore confined to electrocardiographic and vectorcardiographic leads which record the vertical or Y component of the cardiac vector Thus the precordial leads of the electrocardiogram and the horizontal projection of the vectorcardiogram do not show diagnostic changes while leads II III and aVF and the frontal and sagittal projections of the vectorcardiogram typically display the abnormalities diagnostic of diaphragmatic infarction While it may sometimes happen in diaphragmatic infarction that leads V_1 and V_2 record QRS deflections of increased resultant positivity depressed S-T segments and tall upright T waves these changes are usually related to coexisting infarction of the strictly posterior or the posterolateral aspect of the left ventricle

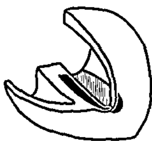
Although infarctions involving the inferior wall of the left ventricle continue to be referred to by many as *posterior* infarctions this designation is being supplanted gradually by the terms *diaphragmatic* or *inferior* both of which are used interchangeably in this text The term "posterior is reserved by the authors specifically for infarctions producing unbalanced forces directed anteriorly

The Instantaneous VA Vectors

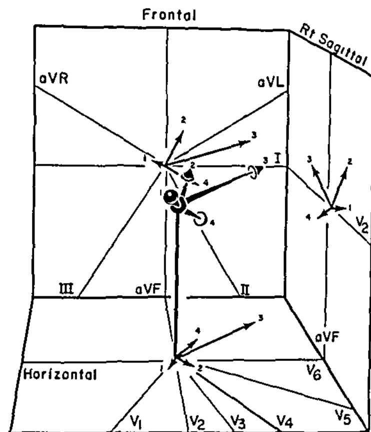
In describing the instantaneous VA vectors in diaphragmatic infarction (see also Fig 181) only the vertical and transverse components of the vectors will be considered since typically the anteroposterior component of the instantaneous vectors is not altered by the infarction and is therefore the same as in the normal electrocardiogram Accordingly in the discussion the instantaneous VA vectors will only be related to the lead deflections recorded in leads II III and aVF which are the diagnostic leads in diaphragmatic myocardial infarction

0 01 Second VA Vector

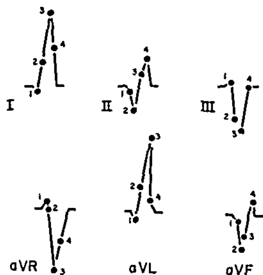
Although posterobasal septal involvement in diaphragmatic infarction is not uncommon the initial septal forces usually retain their normal magnitude



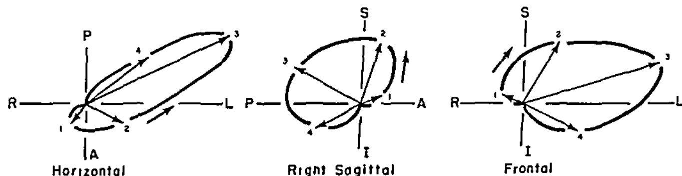
A Diaphragmatic Myocardial Infarction



B Instantaneous VA Vectors in Diaphragmatic Myocardial Infarction



C QRS Deflections Projected on Scalar Leads



D Planar QRS Loops in Diaphragmatic Myocardial Infarction

ever this is the equivalent of a QS deflection inasmuch as the

tively to

and direction to the right and superiorly (or occasionally inferiorly)

Leads II III and aVF —Beginning downstroke of a Q wave (or a small initial R wave if the 0 01 second VA vector is directed inferiorly)

0 02- and 0 04-Second VA Vectors

Transmural activation of the diaphragmatic wall of the left ventricle occupies approximately the period between 0 02 and 0 04 second of the QRS interval during which time this region of the heart normally produces electrical forces directed to the left and inferiorly In diaphragmatic infarction these leftward and inferiorly directed forces are subtracted from the balance of cardiac forces existing at each instant of

but opposite direction the latter being represented by an infarction vector directed superiorly and slightly to the right As previously explained the postinfarction 0 02 and 0 04 second vectors are resultants of the corresponding preinfarction vectors and the infarction vector

Thus in diaphragmatic myocardial infarction the 0 02 second VA vector is displaced from its normal position (inferior to the left, and slightly anterior) to a position far superior and more medial or slightly to the right Similarly there is usually upward displacement of the 0 04 second VA vector in this type of infarction Although the 0 04 second VA vector ordinarily remains the maximal mean instantaneous vector it generally does not extend as far superior as does the 0 02-second VA vector

Leads II III and aVF —Deep wide Q waves (if the 0 01 second VA vector and the 0 02 and 0 04 second vectors are all oriented superiorly) or wide S waves following small initial R waves (if the 0 01 second vector is directed inferiorly and the 0 02 and 0 04-second vectors superiorly)

0 06-Second VA Vector

If the basal diaphragmatic wall, which is activated relatively late in the QRS interval, is not involved

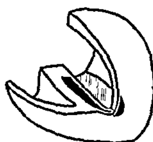
tend to be oriented more to the left and somewhat inferiorly or less superiorly than the preceding instantaneous vectors

Leads II III and aVF —Depending on the location of the 0 06-second VA vector—and this can be variable—the vector projects a small terminal R wave or a terminal S wave on one or more of leads II III and aVF When these leads record a terminal R wave the latter is smaller than normal because the 0 04 second vector in diaphragmatic infarction is displaced superiorly and therefore contributes little or nothing to the formation of the terminal R wave On the other hand frequently *there is displacement of the late instantaneous vectors in much the same direction as the vectors appearing during the first 0 04 second of the QRS interval possibly reflecting extension of the infarction to the inferobasal area of the left ventricle In such an instance the 0 06-second VA vector and subsequent instantaneous vectors are directed almost as markedly superiorly as are the preceding vectors*

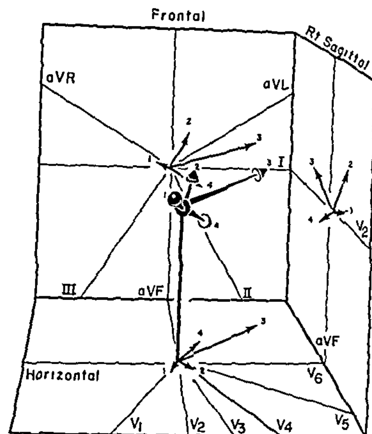
Before summarizing the various configurations of the QRS deflections recorded in leads II III and aVF in *diaphragmatic myocardial infarction* the fact should be re-emphasized that typically in this type of infarction the 0 01 second VA vector is normal in direction and magnitude Thus if before infarction the 0 01 second vector was situated superiorly then it remains so after infarction In this event the 0 01 to 0 04 second VA vectors all project negative potentials in the form of wide deep Q waves on leads II III and aVF On the other hand if it should so happen that the preinfarction 0 01 second VA vector (and therefore the corresponding postinfarction vector) is directed inferiorly then lead aVF and/or lead III record small initial R waves followed by S waves of larger size Often when this is the case the 0 06-second VA vector projects a terminal R on lead aVF and/or lead III so that an rSR or rSr′ complex is registered instead of an rS deflection When the 0 01 second VA vector is located between +150 and 180 in the frontal *reference frame only lead II* records a Q *wave while leads III* and aVF show initial small R waves Occasionally when the limb leads show apparent left axis deviation of Â QRS with leads III and aVF recording rS deflections the presence of a QS or qrS deflection in lead II may provide the clue to the correct diagnosis of diaphragmatic myocardial infarction *since this circumstance does not usually happen in left* axis deviation in the *absence of infarction* (but may occur occasionally in chronic pulmonary heart disease)

QRS Criteria for Diagnosis

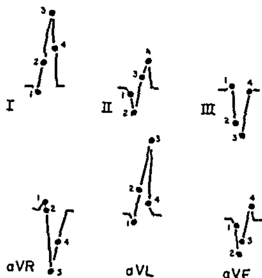
Ordinarily the electrocardiographic recognition of an acute diaphragmatic myocardial infarction pre



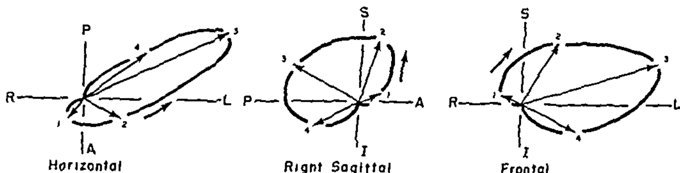
A Diaphragmatic Myocardial Infarction



B Instantaneous VA Vectors in Diaphragmatic Myocardial Infarction



C QRS Deflections Projected on Scalar Leads



D Planar QRS Loops in Diaphragmatic Myocardial Infarction

tion of the ically inert tantaneous es on leads s not affected by the infarction and in the example depicted happens to project on the positive half of the axis of derivation of lead III this lead registers a minute R wave followed by a wide S wave However this is the equivalent of a QS deflection Inasmuch as the abnormal displacement of the VA vectors occurs primarily in a superior direction no diagnostic abnormalities appear in the precordial leads in diaphragmatic infarction Note in **D** that the efferent limbs of the sagittal and frontal loops can be seen to be displaced abnormally superiorly which in turn causes a reversed direction of inscription of these loops on the other hand the horizontal QRS loop is relatively normal in appearance *The schematic loops are quite characteristic for diaphragmatic infarction*

dence of acute infarction plus vectorcardiographic abnormalities unmistakably indicative of diaphragmatic infarction) The qualifying terms "diagnostic" or abnormal when applied *hereafter* to Q waves appearing in leads II III and aVF are meant to signify that the Q wave in *question satisfy* both width and depth *criteria described* above The frequency of diagnostic *Q waves in* leads II III and aVF in our 45 cases of acute or old *diaphragmatic* myocardial infarction is summarized as follow

	% OF ELECTROCARDIOGRAMS
No diagnostic Q waves in leads II III and aVF	36
Diagnostic Q waves in all three leads	12
Diagnostic Q waves in lead III only	20
Diagnostic Q waves in lead aVF only	8
(Note o electrocardiogram displayed diagnostic Q waves in lead II alone)	
Diagnostic Q waves in leads III and aVF	24
Relative frequency of diagnostic Q waves in the respective leads	
Diagnostic Q waves in lead III	56
Diagnostic Q waves in lead aVF	44
Diagnostic Q waves in lead II	12

Although the above percentages would seem to imply that lead III is the lead of diagnostic preference in diaphragmatic myocardial infarction this is not entirely *true* for lead III is more prone than either of the other two leads to yield false positive interpretations of infarction This fact *holds* true especially if the electrocardiographer *requires* that the Q *waves in lead III* satisfy only one or the other of the width *and* depth criteria *to be considered* diagnostic *To illustrate this point, we* shall turn once again to our own observations in a series of electrocardiograms recorded from patients of varying ages without *evidence of* cardiac disease Diagnostic Q waves fulfilling both width and depth criteria of abnormality were noted in lead III alone in 42 of the electrocardiograms of these patients, while Q waves in lead III *satisfying one of the criteria of abnormality* and almost fulfilling the other criterion were observed in 12% of the *records* Aside from the foregoing findings in normal electrocardiograms it is well established that in the absence of infarction diagnostic Q waves may frequently occur in lead III and less *frequently* in lead aVF *in* chronic pulmonary emphysema with or without cor pulmonale in acute cor pulmonale or right bundle branch block and even occasionally in left ventricular hypertrophy From the over all standpoint both of diagnostic sensitivity and reliability if any lead deserves to be considered the *preferred* diagnostic lead in diaphragmatic myocardial infarction it is probably lead aVF The explanation underlying this preference is presented in the following paragraphs

The ... III ... matic ... direction is ... tion vector in this type of *infarction* and the axis ... derivation of leads II and III As will *be recalled* the infarction vector in diaphragmatic myocardial infarction causes *superior* displacement of the postinfarction mean instantaneous QRS spatial vectors associated with little or no abnormal deviation of the vectors along the transverse axis Obviously the abnormal superiorly directed unbalanced forces created by the infarction are most accurately recorded by a vertic ... for all intents and purposes only ... tor T ... aVF ... and III are rotated at least 30 to either side of the vertical axis implies that these leads respond not only to the vertical component but also to the transverse component of a given cardiac vector The possible inaccuracies which can be introduced by the foregoing deficiencies of leads II and III can be exemplified by the following

1 Any mean instantaneous QRS vectors situated in the +150 to 160 segment of the frontal reference frame project negative voltages on lead II even though situated inferiorly and the same holds ... in the case of lead III for vectors located in the 0 to

... *vectors in question*

2 Conversely superiorly situated mean instantaneous QRS vectors which happen to lie in the 0 to −30 segment of the frontal reference frame project positive voltages on lead II similarly superiorly located vectors lying in the 160 to −150 segment of the frontal reference frame likewise *project positive* voltages on lead III *In either case* lead aVF registers negative voltages signifying that the vectors are truly oriented superiorly

3 In diaphragmatic myocardial infarction the mean instantaneous QRS *vectors* which are displaced *by the infarction vector are those appearing between* 0 015 and 0 04 *second—that is* vectors usually situ*ated to the* left. Consequently the corresponding

sents little difficulty because sooner or later in most instances serial electrocardiograms not only will show diagnostic QRS abnormalities but—of equal or even greater importance—will also display characteristic evolutionary changes in the S–T segments and T waves in leads II III and aVF (Fig 185) Quite an other story is the healed or old diaphragmatic myocardial infarction which so often poses a knotty diagnostic problem for the electrocardiographer since his decision as to the presence or absence of infarction rests solely on a correct evaluation of the QRS deflections or Q waves (if present) in the diagnostic leads (The same holds equally true for healed infarction involving other aspects of the left ventricular wall) To say the least the QRS residuals of healed diaphragmatic myocardial infarction are quite equivocal at times Therefore to do justice to the importance of this problem obviously the QRS criteria for diagnosing acute or old diaphragmatic myocardial infarction merit special attention

From the very outset of the discussion the fact must be kept in mind that the most reliable criteria of QRS abnormality in leads II III and aVF (Q $\geqq$0 04 second and >25% of the following R wave amplitude) are burdened with an inherent handicap i e they pertain solely to *Q wave abnormality* in diaphragmatic infarction Thus the criteria ignore the significant percentage of electrocardiograms (about 15% in the series studied by the authors of this text) which exhibit rS or rSR deflections in leads II III and aVF as the only residuals of infarction Some idea of the value of the aforementioned criteria in recognizing old diaphragmatic myocardial infarction can be derived from our observations in 45 cases of acute and old infarction (all cases having either unequivocal clinical histories of old infarction or ancillary evi

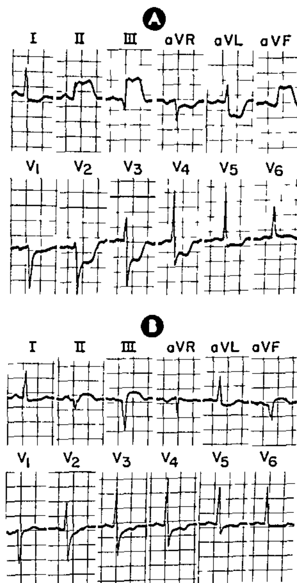
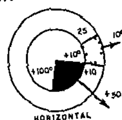
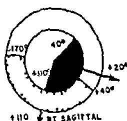
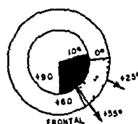


Fig 185 —Evolution of electrocardiographic abnormalities diagnostic of acute diaphragmatic myocardial infarction The findings in the initial record (**A**) are typical of the early phase of diaphragmatic myocardial infarction and consist of marked S–T segment elevation and upright T waves in leads II III and aVF Although a diagnostic Q wave is present in lead III significant Q waves have not yet appeared in leads II and aVF The S–T segment depression in leads V through V may possibly represent anterior subendocardial myocardial injury but is far more likely to be reciprocal to the subepicardial injury involving the posterior or infra atrial wall of the left ventricle The second electrocardiogram (**B**) was recorded 24 hours later and differs from the preceding as follows the S–T segment elevation in leads II III and aVF and the S–T segment depression in leads V_1 through V are less marked than before but inverted T waves are now present in these leads (with the exception of leads V and V) Note that leads III and aVF now display QS deflections and lead II a Qr complex

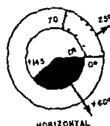
Normal (60 Cases)

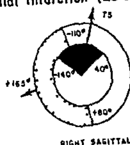


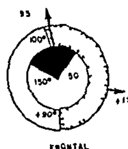




Diaphragmatic Myocardial Infarction (25 Cases)







MEAN .02 SEC INSTANTANEOUS QRS VECTOR OF PLANAR QRS LOOP

MAXIMAL MEAN INSTANTANEOUS QRS VECTOR OF PLANAR QRS LOOP

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tors of t
orientation of a given instantaneous v to is

deviated superiorly on occasions As a general rule

the orientation and perhaps the relative magnitude of the 0 02 second instantaneous QRS vector in the vectorcardiograms of patients suspected of infarction The data we obtained from analyses of the vectorcardiograms of a series of patients with diaphragmatic infarction is summarized in Table 25 and Figure 186

FRONTAL QRS LOOP —During the first 0 02–0 04 second of the QRS interval the frontal QRS loop in diaphragmatic myocardial infarction is characterized

orly and then the efferent limb is inscribed to the left and abnormally superiorly The long axis of the loop (corresponding to the maximal mean instantaneous QRS vector in the frontal projection) may

TABLE 25 —ORIENTATION OF THE MEAN 0 02 SECOND AND MAXIMAL MEAN INSTANTANEOUS VECTORS OF THE QRS sÊ LOOP IN DIAPHRAGMATIC INFARCTION

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	A	Usual Range	Extreme Range	A	Usual Range	Extreme Range	A	Usual Range
Mean 0 02-second instantaneous QRS vector	0 to +145	+60	+10 to +100	−140 to −40	−75	−90 to −40	−150 to −50	−95	−150 to −50
Maximal mean instantaneous QRS vector	−70 to 0	−25	−45 to 0	+80 to −110	+165	+130 to −140	−100 to +90	+15	−40 to +30

Usual range = range in 85% of cases.

postinfarction vectors usually lie abnormally superiorly and to the left and tend on the whole to project deceptively small negative voltages on lead II because of their leftward orientation By the same token in diaphragmatic lateral myocardial infarction a common type of combined infarction the mean instantaneous QRS vectors are displaced not only superiorly but also to the right In this instance lead III tends to register deceptively small negative deflections

Other criteria like the R_{III} peak interval (the interval from onset of the QRS to the peak of the R wave in lead III ≧ 0 06 second) for example whether used singly or in combination with the Q width and depth criteria in lead aVF have not increased the diagnostic accuracy of the electrocardiogram in diaphragmatic infarction This problem has been approached somewhat differently by Grant and others who utilize vector projection methods to determine the orientation of initial and early QRS vectors in diaphragmatic infarction The method used by Grant to construct the mean 0 04 second QRS spatial vector from the electrocardiogram was described earlier in Chapter 3 In the large series of normal subjects studied by Grant the mean 0 04 second QRS spatial vector was found to be oriented invariably to the left posteriorly and inferiorly while in diaphragmatic myocardial infarction this vector was usually directed markedly superiorly rather than inferiorly Vector methods have been applied to the diagnosis of diaphragmatic infarction in a somewhat different way by Pearce and Chapman who have recorded simultaneous leads aVF and V_B on a twin channel electrocardiograph Lead V_B was obtained by placing the exploring chest electrode just to the left of the seventh thoracic vertebra The 0 01 and 0 02 second sagittal vectors were constructed by measuring the deflections at these intervals in the two leads and the measurements were then utilized as the tangent of the angle of the vector According to Pearce and Chapman almost all of the autopsy proved diaphragmatic infarcts in their series had 0 02 second sagittal vectors lying between −65 and −150 The patients with prominent Q waves in lead aVF who did not show diaphragmatic infarction at postmortem uniformly had 0 02 second sagittal vectors situated inferior to −65 Although there was a significant number of false positive interpretations Pearce and Chapman found that these could be eliminated by combining the following two criteria (*a*) a 0 02 second sagittal vector situated between −65 and −150 and (*b*) a Q_{VF} interval of 0 04 second or longer

Probably the most useful adjunct to the electrocardiogram in the diagnosis of inferior or diaphragmatic infarction is the vectorcardiogram and this is particularly true in those cases in which leads III and aVF display rS or rSR deflections

Vectorcardiographic Findings

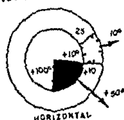
Since the unbalanced forces created by infarction of the inferior aspect of the left ventricle are directed superiorly away from the electrically inert muscle the resulting disturbance of the balance of cardiac forces occurs along the Y or vertical axis Consequently the vectorcardiographic abnormalities diagnostic of diaphragmatic infarction are to be sought in the two vectorcardiographic projections having a component lead which responds to the Y component of the cardiac vector—namely the sagittal and frontal projections Nevertheless the horizontal projection of the QRS sÊ loop is included in the following description of the QRS sÊ loop findings in diaphragmatic infarction

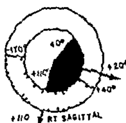
Horizontal QRS loop —The horizontal QRS loop in diaphragmatic myocardial infarction ordinarily presents essentially a normal appearance being counterclockwise inscribed and showing a normal initial deflection to the right and anteriorly The mean 0 02 second instantaneous vector of the horizontal QRS loop is normally oriented but the range of variation and average orientation of the maximal mean instantaneous vector of the loop tend to be situated farther posteriorly than the corresponding vector in the nor

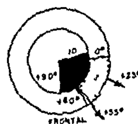
ities in its configuration

Right sagittal QRS loop —The chief diagnostic abnormality of the sagittal QRS loop and the feature most consistently present in diaphragmatic infarction is that during the first 0 02–0 04 second of the QRS interval the loop is written markedly superiorly and usually slightly anteriorly The pronounced superior displacement of the initial and early instantaneous vectors frequently causes counterclockwise inscription of the sagittal loop in its entirety or counterclockwise inscription of the proximal component of a figure of eight sagittal loop Counterclockwise inscription was observed in over two thirds of the vectorcardiograms which we considered to be diagnostic of diaphragmatic infarction Although the most striking evidence of diaphragmatic infarction in terms of superior displacement of the instantaneous vectors is usually seen in the initial and early portions of the QRS sÊ loop the efferent limb long axis of the sagittal QRS loop and sometimes the afferent limb may one or all be

Normal (60 Cases)

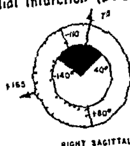


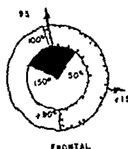




Diaphragmatic Myocardial Infarction (25 Cases)







MEAN .02 SEC INSTANTANEOUS QRS VECTOR OF PLANAR QRS LOOP

MAXIMAL MEAN INSTANTANEOUS QRS VECTOR OF PLANAR QRS LOOP

Fig 186 —Range of variation and average orientation of the mean 0 02-second and maximal mean instantaneous vectors of the planar QRS loops in normal subjects and in patients with diaphragmatic myocardial infarction The average orientation of a given instantaneous vector is indicated by a vector arrow

deviated superiorly on occasions As a general rule [illegible]

[illegible] the orientation and perhaps the relative magnitude of the 0 02 second instantaneous QRS vector in the vectorcardiograms of patients suspected of infarction. The data we obtained from analyses of the vectorcardiograms of a series of patients with diaphragmatic infarction is summarized in Table 25 and Figure 186

Frontal QRS Loop —During the first 0 02–0 04 second of the QRS interval the frontal QRS loop in diaphragmatic myocardial infarction is characterized by a prominent deflection extending abnormally superiorly Initially the loop is written slightly to the right and usually superiorly although occasionally inferiorly and then the efferent limb is inscribed to the left and abnormally superiorly The long axis of the loop (corresponding to the maximal mean instantaneous QRS vector in the frontal projection) may

TABLE 2? —Orientation of the Mean 0 02-Second and Maximal Mean Instantaneous Vectors of the QRS sE Loop in Diaphragmatic Infarction

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range *	Extreme Range	Av	Usual Range
Mean 0 02-second instantaneous QRS vector	0 to +145	+60	+10 to +100	−140 to −40	[illegible]	−90 to −40	−150 to −50	−85	−130 to −50
Maximal mean instantaneous QRS vector	−70 to 0	−25	−45 to 0	+80 to −110	+165	+130 to −140	−100 to +90°	+15	−40 to +30

* Usual range = range in 8 % of cases

sometimes lie above the 0° axis in diaphragmatic myocardial infarction although in our cases the average orientation of the frontal QRS loop was essentially the same as that in normal subjects After the appearance of the maximal instantaneous vector the afferent limb returns inferiorly in most instances to complete inscription of the loop in a clockwise direction However sometimes the afferent limb may be situated above the efferent limb the frontal loop in this event having a counterclockwise direction of inscription As a general rule when the frontal QRS loop in diaphragmatic myocardial infarction is written in a counterclockwise direction the efferent limb of the loop is not only displaced superiorly but tends to be inferiorly concave that is to be bowed upward in its midportion In addition frontal QRS loops with a figure of eight configuration are often observed in diaphragmatic myocardial infarction the proximal loop of the eight having a clockwise direction of inscription and the distal component a counterclockwise direction of inscription This variant type of frontal QRS loop configuration was observed by us more frequently in old healed diaphragmatic myocardial infarction than in more recent infarctions

As Table 25 indicates the orientation of the 0 02 second instantaneous vector of the frontal QRS loop would seem to be a better point of distinction between the normal frontal loop and the loop in diaphragmatic myocardial infarction than the orientation of the maximal instantaneous QRS vector or long axis of the

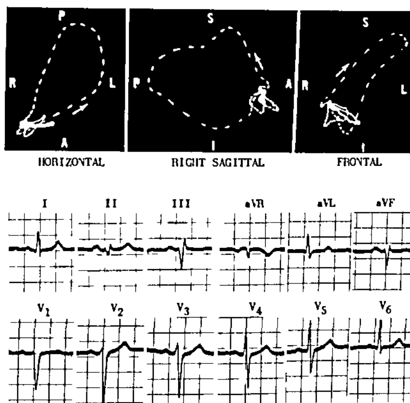


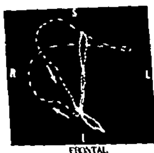
Fig 187 —Electrocardiographic and vectorcardiographic findings in old or healed diaphragmatic myocardial infarction

If one adheres to the conventional empiric criteria for the electrocardiographic diagnosis of diaphragmatic infarction this diagnosis could not be made in the electrocardiogram in this figure because of the fact that leads II III and aVF register RSR deflections This electrocardiogram is a good case in point illustrating the advantages of the vector approach to the electrocardiogram On calculating the mean 0 04 second QRS spatial vector from the electrocardiogram the vector will be found to lie at −50 in the frontal reference frame in contrast with the inferior orientation of the normal mean 0 04 second QRS spatial vector Thus the electrocardiographic diagnosis of diaphragmatic infarction can be [illegible] (The limitations of this method are discussed in Chapter 7)

leads II III and aVF of [illegible] the superior displacement of subsequent [illegible] The in
loop accounts for the pr[illegible] ctorcar
farction would certainly [illegible]
diogram and the T sE lo[illegible]







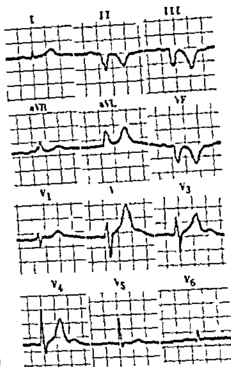


Fig 188 —Electrocardiographic and vectorcardiographic findings in a recent diaphragmatic anterolateral infarction

In the electrocardiogram the QRS duration is almost 0 12 second QS deflections and deeply inverted T waves are recorded by leads II III and aVF The QRS deflections in leads I and V are of low amplitude, but the small Q waves in these leads are nondiagnostic of infarction. The width of the R wave in Lead V is between 0 02 and 0 03 second, and so the R wave in V cannot be considered indicative of anterolateral infarction

The vectorcardiographic abnormalities diagnostic of diaphragmatic infarction are obvious and will not be described. Note the elongated, large and superiorly directed T sE loop indicative of diaphragmatic ischemia The vectorcardiographic findings diagnostic of coexisting anterolateral infarction are as follows the mean 0 02-second instantaneous QRS vectors in the frontal and horizontal QRS loops are directed much farther to the right and anteriorly (as well as superiorly) than normally and the efferent limbs of the horizontal and frontal QRS loops are displaced to the right or medially superiorly and posteriorly Note the reversed direction of inscription of the early deflection of the horizontal QRS loop The prolonged QRS interval may reflect peri infarction block (discussed on p 335 Chapter 21)

loop In normal subjects we found the 0 02 second vector in the frontal plane to be located between 0 and +60 in three fourths of the cases while in most cases of diaphragmatic myocardial infarction the 0 02 second vector was situated between −40 and −140 The average orientation of the mean 0 02 second instantaneous vector of the frontal QRS loop in diaphragmatic myocardial infarction was found by us to be −95 while the orientation of the vector in normal subjects averaged +55 Over 80% of the frontal loops in the infarction series were inscribed in clockwise direction a considerable increase in the frequency of this finding as compared with normals

S–T Vector and Ventricular Repolarization

In acute diaphragmatic myocardial infarction there is present, for reasons previously cited an S–T vector which is directed toward the effective electrical site of subepicardial injury—that is inferiorly and this vector projects elevated S–T segments on leads II III and aVF The corresponding abnormality of the QRS sE loop of the vectorcardiogram consists of a shift of the terminus of the loop inferiorly a line drawn from the loops point of origin to its terminus indicating the direction and magnitude of the S–T vector

Subsequently as the strong injury field subsides during the evolution of a diaphragmatic myocardial infarction the changes due to transmural ischemia at the site of infarction make their appearance in the electrocardiogram and vectorcardiogram The local reversal in the direction of repolarization consequent to the infarction induced delay in subepicardial myocardial recovery tends to rotate the instantaneous T vectors away from the effective electrical site of the

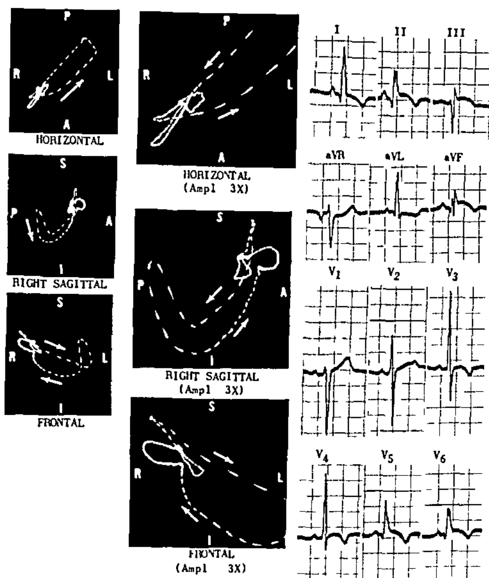


Fig 189 —Electrocardiographic and vectorcardiographic findings at an early stage in the evolution of an acute diaphragmatic myocardial infarction There is S-T segment elevation indicative of diaphragmatic lateral subepicardial injury in leads II III aVF V and V of the electrocardiogram and in the same leads inverted T waves of transmural ischemia are also present The Q waves in leads II and aVF are as yet insignificant while lead III registers an rSr deflection Thus unequivocal evidence of actual muscle necrosis in the electrocardiogram has not appeared up to this point In the vectorcardiogram the terminus of the QRS sÊ loop is displaced anteriorly inferiorly and somewhat to the left indicating a similarly directed S-T vector while the T sÊ loop is oriented anteriorly to the right and somewhat superiorly The right sagittal QRS loop exhibits a relatively prominent markedly superiorly directed early deflection with a 0 02 second mean instantaneous vector lying at about −80 In addition there is a reversed direction of inscrip

infarction—that is superiorly and somewhat to the r ... T vectors project on the negative
l

... T waves These findings in the electrocardiogram ... paralleled by the changes in the T sE loop of the vectorcardiogram For example in recent diaphragmatic infarction the T sE loop is usually directed superiorly and slightly to the right and anteriorly it tends to be greatly elongated and the efferent and afferent limbs of the loop may be inscribed at nearly the same rate (the time dashes being evenly spaced throughout) If the infarction is relatively old the characteristics of the T sE loop are much more variable the loop may have a normal appearance and variable orientation it may be small round and oriented superiorly or away from QRS sE loop or it may have a normal appearance but discordant orientation with respect to the long axis of the QRS sE loop (Figs 187–189)

POSTEROLATERAL INFARCTION

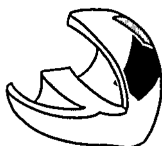
The second type of inferoposterior infarction posterolateral infarction has received far less attention in the past than any of the other infarction patterns which have so far been described In fact, the frequent, although not invariable association of diaphragmatic and posterolateral infarctions led in previous years to grouping these two infarction patterns together under the common designation of posterior infarction Since the latter term was also applied to diaphragmatic infarction unaccompanied by posterolateral involvement this naturally resulted in considerable confusion in terminology and meaning (and tended to obscure the separate identity of posterolateral infarctions)

It will be remembered that during the description of the anterior myocardial infarctions the point was made that lateral infarctions are for all intents and purposes of two types anterolateral infarction and posterolateral infarction The *anterolateral* type of infarction is so designated because its electrical location is in the anterolateral wall of the left ventricle Thus the infarction vector in anterolateral infarction is directed to the right and posteriorly (see preceding chapter) In contrast *posterolateral* infarctions are placed in terms of their effective electrical position in the posterolateral or posterobasal wall of the left ventricle The infarction vector representing the unbalanced forces created by the infarction is directed to the right and anteriorly

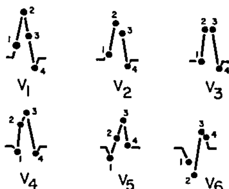
In theory at least one would expect the unbalanced forces resulting from posterolateral infarction to affect the electrocardiogram and vectorcardiogram during the time this region of the left ventricle normally undergoes activation—that is from about 0 04 second after onset of the QRS interval until the end of this period As will be indicated later alterations of the terminal portion of the QRS sE loop of the vectorcardiogram and of the QRS deflection of the electrocardiogram in fact do frequently occur in posterolateral infarction although they have not been extensively studied as yet The electrocardiographic abnormalities generally accepted as being diagnostic of posterolateral infarction (tall wide R wave in lead V_1 and deep wide Q wave in leads V_6 and or V_7) involve the earlier portion of the QRS deflection and therefore appear before activation of posterolateral left ventricular wall In view of this fact it is evident that, in addition to or perhaps instead of the mechanism of the QRS changes of infarction described earlier (Chapter 19) some other mechanism must be implicated to account for the early QRS abnormalities in posterolateral infarction and this may well hold true for infarctions in other locations Grant has summarized the problem of explaining the Q waves of infarction in the following way " ... whatever the anatomic location of the infarct its electrical mechanism must be such that the very first part of the myocardium to generate electrical activity of sufficient magnitude to be recorded at the body surface is involved by the infarction process" He then goes on to speculate that the early stages of ventricular depolarization being a nearly instantaneous process it is possible the concept of conduction relationships which could cause infarcts in various regions of the heart to be referred electrically to more proximate regions along the anterior and posterior subdivisions of the left bundle branch network" However for the sake of simplicity it will be assumed in this text that the QRS abnormalities in posterolateral infarction merely reflect a shift in the balance of cardiac forces resulting from loss of forces directed posteriorly and to the left

The Instantaneous VA Vectors

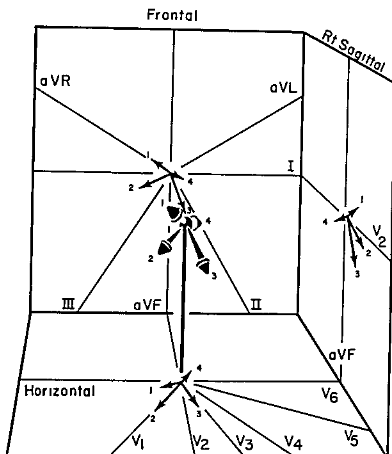
In the absence of concomitant diaphragmatic infarction the unbalanced forces created by posterolateral infarction have two directional components a



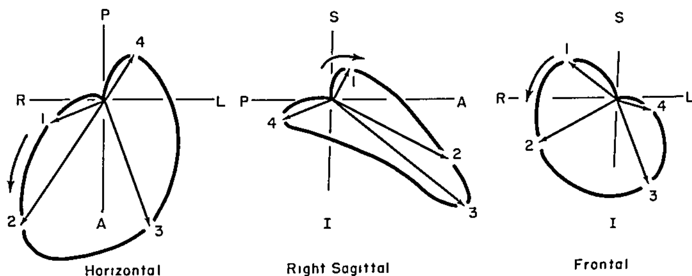
A Posterolateral Myocardial Infarction



C QRS Deflections Projected on Scalar Leads



B Instantaneous VA Vectors in Posterolateral Myocardial Infarction



D Planar QRS Loops in Posterolateral Myocardial Infarction

Fig 190 –Inst
infarction is depi
forces directed to
2 and 3 are deviat
derived from the
Note that just as
R wave Lead V

rightward transverse component (X) and an anterior sagittal (Z) component Typically the balance of cardiac forces is not altered along the vertical (Y) axis in posterolateral infarction Thus in describing the VA vectors in the following paragraphs reference will be made only to the orientation of the vectors along the X and Z axes For the same reason in relating the instantaneous vectors to the electrocardiogram and vectorcardiogram (see Fig 190) the precordial leads of the electrocardiogram and the horizontal projection of the vectorcardiogram will be emphasized for the most part When diaphragmatic and posterolateral infarctions occur in combination the infarction vector is directed not only to the right and anteriorly but also markedly superiorly The presence of the diaphragmatic myocardial infarction does not, however alter the features of the posterolateral infarction in the precordial electrocardiographic leads or in the horizontal vectorcardiogram but simply causes additional abnormalities (already described) to appear in limb leads II III and aVF and in the frontal and sagittal projections of the vectorcardiogram

0 01 Second VA Vector

For reasons that are not clear at present posterolateral infarction behaves electrically first as if its electrical location were in the posterolateral or posterobasal wall of the left ventricle and secondly as if this region of the left ventricle were activated during the first 0 04 second of the QRS interval rather than later as is actually the case In other words the infarction vector in posterolateral infarction makes its appearance early in the QRS interval and is directed to the right and somewhat anteriorly While the basal portion of the septum may sometimes be involved in posterolateral infarction in most cases initial septal depolarization from left to right is not disturbed so that the 0 01 second VA vector is directed to the right and anteriorly just as normally is the case projecting the following deflections on the precordial leads and leads I and aVL

Lead V Beginning upstroke of an R wave

Leads V and V and sometimes leads I aVL and V —Beginning downstroke of a Q wave

0 02 Second VA Vector

Although posterobasal left ventricular wall is activated later than 0 02 second after onset of the QRS interval for some reason the unbalanced forces resulting from posterolateral infarction begin to affect the balance of cardiac forces at this time The postinfarction 0 02 second VA vector which is the resultant of the corresponding preinfarction vector (normally of relatively small magnitude and having a leftward and slightly anterior orientation) and the infarction vector is displaced to the right and anteriorly and its magnitude is increased so that the following deflections are written in the electrocardiogram

Lead V_1—*The 0 02 second VA vector coincides* roughly with the peak of the tall R wave in this lead since this vector lies well anteriorly and usually extends farther to the right than any other instantaneous vector

Leads V_5 and V and sometimes leads I aVL and V_4—The 0 02-second VA vector coincides roughly with the nadir of the Q wave in these leads since it usually projects maximally on the negative halves of the axes of derivation of these leads

0 04 Second VA Vector

In a typical posterolateral infarction the 0 04 second VA vector is situated to the left and well anteriorly in contrast with its normal posterior location Occasionally it may even be to the right and anteriorly Whether oriented to the left or right the 0 04 second vector is in most instances located sufficiently anteriorly to project on the positive half of the axis of derivation of lead V_1 that is it is generally situated to the right of +30° in the horizontal reference frame The less marked displacement of the 0 04 second vector in posterolateral infarction in comparison with the 0 02 second VA vector may perhaps be related to the greater magnitude of the preinfarction 0 04 second vector which normally is directed to the left and posteriorly Thus the postinfarction 0 04 second vector tends on the average to conform in terms of its direction more nearly to the direction of the preinfarction 0 04 second vector than to the direction of the infarction vector Consequently the postinfarction 0 04 second vector usually retains its leftward orientation but shifts anteriorly and produces

ar to the right and anteriorly while
id in the horizontal and frontal QRS

the following deflections in the electrocardiogram

Lead V_1 —More often than not the 0 04 second vector projects on the positive half of the axis of derivation of lead V_1 which therefore completes the inscription of a tall R wave of 0 04 second (or longer) duration Less frequently the 0 04 second vector lies far enough to the left to contribute to the downstroke of a terminal S wave in lead V_1

Leads V_6 *and* V *and sometimes leads I aVL and* V_5 —Beginning upstroke of a low R wave

0 06 Second VA and Subsequent Instantaneous Vectors

The 0 06 second VA vector often has much the same orientation in posterolateral infarction as it has normally—that is markedly posterior and slightly to the left However if anything the orientation of the 0 06 second VA vector shows a wider range of variation in posterolateral infarction than its does normally This and certain other dissimilarities between the terminal vectors in the normal subject and in posterolateral infarction will be discussed more fully subsequently

Lead V_1 —Completion of a terminal S wave of varying size which is usually smaller than the R wave in this lead

Leads V_6 *and* V_7 *and sometimes leads I aVL and* V_5 —Completion of low terminal R wave

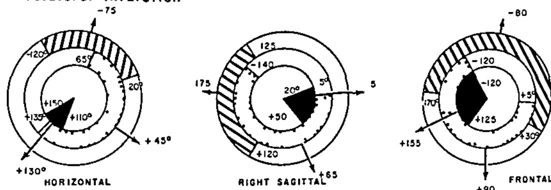
The electrocardiographic criteria for the diagnosis of posterolateral infarction will be outlined after the presentation of the vectorcardiographic findings in this type of infarction

Vectorcardiographic Findings

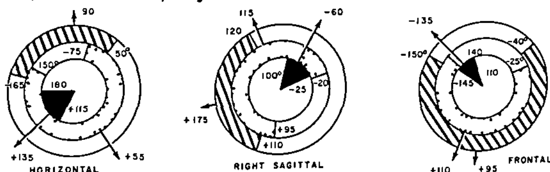
Since the unbalanced forces created by a posterolateral infarction are directed both to the right and anteriorly they produce abnormal changes in the QRS sÊ loop in all three projections of the vectorcardiogram (Fig 191)

Horizontal QRS loop —In the horizontal projec-

Posterolateral Infarction



Posterolateral and Diaphragmatic Infarction



02 SEC MEAN INSTANTANEOUS QRS VECTOR

MAXIMAL MEAN INSTANTANEOUS QRS VECTOR

MAXIMAL TERMINAL MEAN INSTANTANEOUS QRS VECTOR

Fig 191 —Range of variation and average orientation of the 0 02 second mean maximal mean and maximal terminal mean *instantaneous* QRS vectors of the vectorcardiographic QRS sÊ loop in posterolateral and in diaphragmatic posterolateral infarctions

tion the feature which is perhaps most characteristic of and most consistently present in posterolateral infarction is that the QRS loop for the first 0 04 second of the QRS interval is written abnormally far to the right and anteriorly Thus in our series of posterolateral infarctions the mean 0 02-second instantaneous vector as measured from the horizontal QRS loop was oriented on the average along the +130 axis of the horizontal reference frame while the maximal mean instantaneous QRS vector in this projection had an average orientation of +45 In general, the first half of the horizontal QRS loop is located anteriorly and at first to the right and then to the left of the midline and is inscribed in a counterclockwise direction This portion of the QRS loop is therefore responsible for the abnormal Q waves which appear in the left lateral precordial leads and for the tall wide R waves which are recorded in lead V_1 The rest of the horizontal QRS loop is then written in a counterclockwise direction to the left (or sometimes to the right) and posteriorly When the terminal portion of the QRS loop lies in the right posterior quadrant it generally is clockwisely inscribed and causes the QRS loop to have a figure-of-eight configuration The average orientation of the mean 0 06-second instantaneous vector in the authors cases was −75 While the average orientation of the 0 06-second instantaneous vector in posterolateral infarction cited above does not differ significantly from that of the corresponding normal vector exceptions occur in which the terminal 0 06-second vector is located farther to the right and more posteriorly than is observed normally A more common difference between the terminal vectors in posterolateral infarction and those in the normal vectorcardiogram is that the former have a far greater magnitude in many cases than is ever observed normally The reason for this is not apparent

Right sagittal QRS loop —The sagittal QRS loop is clockwise inscribed in many cases of posterolateral infarction, but counterclockwise inscription of the entire loop or of one or the other component of a figure-of eight sagittal loop is not an unusual finding Typically the sagittal loop tends to be somewhat more anteriorly than posteriorly In our cases the mean 0 02-second instantaneous vector was oriented, on the average at −5 the maximal mean instantaneous vector at +65 and the terminal mean 0 06-second instantaneous vector along the −175 axis of the sagittal reference frame

Frontal QRS loop —During the first 0 02–0 04 second of the QRS interval, the QRS sE loop in the frontal projection is generally written far to the right —that is provided the lateral component of the infarction vector is relatively large with respect to the anterior component In this event the rightward extent of the frontal QRS loop projects abnormal Q waves on lead I However if the major displacement of the QRS sE loop takes place in an anterior direction the frontal QRS loop may fail to show diagnostic abnormalities (although it is unusual for the frontal loop in cases such as this to be entirely normal) More often than not the frontal loop is inscribed in a counterclockwise direction although clockwise inscription of the entire loop and in the case of a figure of eight loop clockwise counterclockwise inscription of the initial and terminal components of the loop were found to occur with about equal frequency in our experience. On the average in our cases, the mean 0 02 second instantaneous vector in the frontal projection was found to be situated at about +155 the maximal mean instantaneous vector at about +90 and the terminal mean 0 06-second instantaneous vector at about −80° As a general rule clockwise-inscribed frontal QRS loops frequently show abnormal superior deflections indicative of associated diaphragmatic involvement while counterclockwise-inscribed frontal loops tend to display more striking evidence of lateral wall infarction

Table 26 indicates the range and average orientation of the mean 0 02 second maximal mean and mean 0 06 second instantaneous vectors of the QRS sE loop in each projection in posterolateral infarction with and without concomitant diaphragmatic infarction (Fig 191) It can be seen that diaphragmatic infarction alters the sagittal and frontal instantaneous vectors in a predictable way that is the mean 0 02 second and maximal mean instantaneous vectors are displaced superiorly and to the right and anteriorly

S–T Vector and Ventricular Repolarization

In the vectorcardiographic study of posterolateral infarction our experience has been greater with old infarctions than with acute infarctions The QRS sE

[illegible] However in the vectorcardiog[illegible]

[illegible] orly or anteriorly (the S–T vector being similarly directed) while the T sE loop was large and lozenge sh[illegible]ped and directed to the right and

TABLE 26 —ORIENTATION OF THE MEAN 0 02 SECOND MAXIMAL MEAN AND MEAN 0 06 SECOND INSTANTANEOUS VECTORS OF THE QRS sÊ LOOP IN POSTEROLATERAL MYOCARDIAL INFARCTION WITH AND WITHOUT ACCOMPANYING DIAPHRAGMATIC INFARCTION

	HORIZONTAL			RIGHT SAGITTAL			FRONTAL		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
Posterolateral Infarction without Diaphragmatic Infarction									
Mean 0 02 second instantaneous QRS vector	+110 to +150	+130	+110 to +135	−20 to +50	−5	−20 to 0	+125 to −120	+155	+170 to −120
Maximal mean instantaneous QRS vector	−65 to +135	+45	−15 to +120	−5 to −140	+65	0 to +160	+5 to −120	+90	+5 to +75
Mean 0 06 second instantaneous QRS vector	−120 to −20	−75	−105 to −30	+120 to −125	−175	+140 to −140	−170 to +30	−80	−170 to −40
Posterolateral Infarction with Diaphragmatic Infarction									
Mean 0 02 second instantaneous QRS vector	+115 to 180	+135	+120 to +155	−100 to −25	−60	−70 to −35	−145 to −110	−135	−145 to −130
Maximal mean instantaneous QRS vector	−75 to −150	+55	−20 to −150	+95 to −20	−115	−95 to −20	−25 to −140	+110	+35 to −140
Mean 0 06 second instantaneous QRS vector	−165 to −50	−90	−120 to −50	+110 to −120	+175	+155 to −120	−40 to −150	+95	+35 to −165

Usual range = range in 85% of cases

anteriorly We have found it surprisingly easy to recognize posterior ischemia vectorcardiographically in contrast with the difficulties encountered electrocardiographically

Electrocardiographic Criteria for Diagnosis

QRS Abnormalities

The electrocardiographic abnormalities most characteristic of posterolateral infarction are (Fig 192)

1 *Abnormal Q waves in leads V_6 and/or V_7 and* sometimes in leads I aVL and V_5
2. An R wave in lead V_1 with a duration of 0 04 second or longer
3 Increased amplitude of the R wave in V_1 with R S ratio equal to or greater than 1

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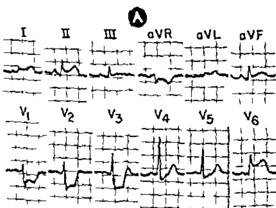
cillary evidence of myocardial infarc

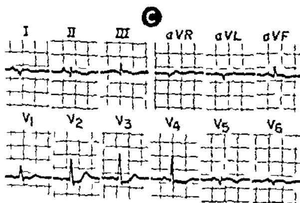
incidence of the above electrocardiographic findings was as follows

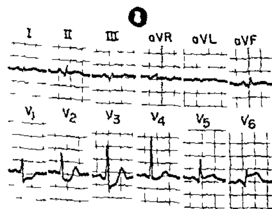
Lead V_1 —In 75% of the cases R/S ≧ 1 and width of R wave ≧ 0 04 second in 25% of cases R/S<1 and width of R wave = 0 03–0 04 second

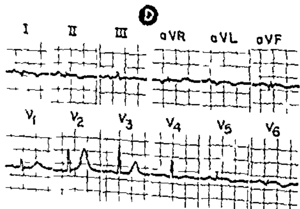
Leads V_6 *and/or* V_7 —Abnormal Q wave in 75% of cases

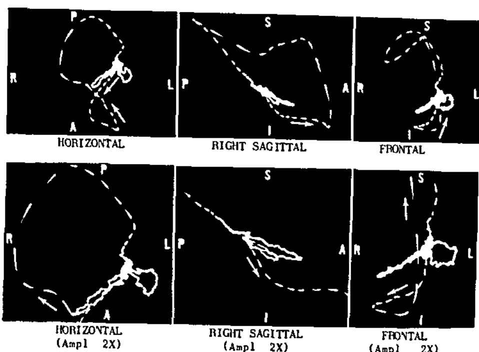
See Criteria of Q Wave Abnormalities in Chapter 18

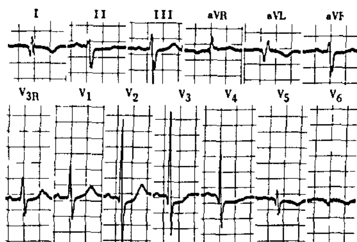












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R ۱

QR

riorly and to the right the sagitt il QRS loop is counterclockwise inscribed at first anteriorly and later posteriorly while the frontal QRS loop is written almost entirely along the vertical axis The T sÊ loop is directed anteriorly and to the right indicating posterolateral ischemia

Lead I —Abnorm il Q wave in 50% of cases

Lead aVL —Abnormal Q wave in 15% of cases

Leads I aVL V_6 and V_7 —Equivocal normal or absent Q waves in all leads in 25% of cases

Leads II III and aVF —Abnormal Q waves in 45% of cases (i e 45% of cases of posterolateral infarction selected by the authors for study evidenced associated diaphragmatic myocardial infarction)

It has been our experience that diagnostic findings in lead V_1 in posterolateral infarction tend on the average to be more prominent and more consistently present than those occurring in lead V_6 although it is equally true that the findings in lead V_1 are less specific for posterolateral infarction than those in lead V_6 For example in some normal individuals with normal vectorcardiograms the R/S ratio in lead V_1 may exceed 1 and the width of the R wave occasionally may approach or equal 0 04 second

As Grishman has pointed out the electrocardiographic features of healed posterolateral infarction

may resemble quite closely those of the so-called *classic* or *uncommon* type of right bundle branch block or may simulate right ventricular hypertrophy. In differentiating these conditions one from the other and in establishing the diagnosis of posterolateral infarction the vectorcardiogram possesses unequivocal diagnostic advantages over the electrocardiogram.

S-T Segment and T Wave Changes

In the subepicardial myocardial injury phase of acute posterolateral infarction an S-T vector points toward the effective site of injury—that is posteriorly and to the left. Thus the S-T segments in leads V_7 and usually V_8 are upwardly displaced while leads V_{3R}, V_1 and V which are oriented to the negative aspect of the injury vector record depressed S-T

overlie the infarction. Whether one can attribute the pronounced S-T segment depression in leads V_1 and V in acute posterolateral infarction to the additive effects of posterior subepicardial injury and anterior subendocardial injury is conjectural.

Posterolateral transmural ischemia causes the instantaneous T vectors to rotate anteriorly and to the right. Inverted T waves are thus recorded in leads V_8 and V_7 but the remaining routine precordial leads register upright T waves usually larger. As a rule the right precordial leads in posterolateral infarction display QRS, S-T and T abnormalities which are inverted mirror images of the abnormalities in left posterior back leads. For example the tall and wide R waves recorded in lead V_1 correspond to the deep and wide Q waves recorded in lead V_8, the depressed S-T segments in lead V_1 are reciprocally related to the elevated S-T segments in lead V_8, and the tall upright T waves registered in lead V_1 correspond to the deeply inverted T waves present in lead V_8. The pattern for the evolution of the S-T segment and T wave abnormalities in posterolateral infarction is the same pattern as for myocardial infarction (see Chapter 18 and Figs 193–195).

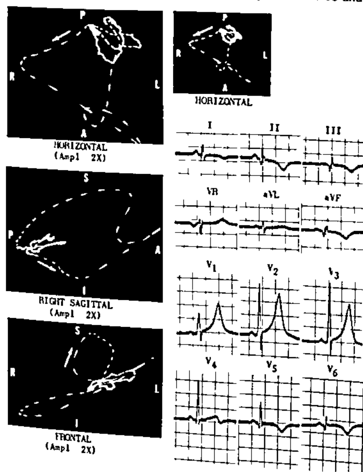


Fig 194 —Electrocardiographic and vectorcardiographic findings in recent posterolateral myocardial infarction.

The electrocardiogram shows an abnormal Q wave in lead V, a tall wide initial R wave in lead V_1, tall upright T waves in leads V through V and inverted T waves in leads I, II, III, aVF, V and V. These findings are all compatible with posterolateral myocardial infarction and diaphragmatic posterolateral ischemia.

In the vectorcardiogram the QRS sÊ loop displays an early deflection which extends abnormally far to the right and anteriorly. This signifies the presence of posterolateral myocardial infarction. There is a large S-T vector directed to the left and posteriorly best demonstrated in the horizontal projection (as evidenced by the leftward and posterior displacement of the terminus of the QRS loop). This finding is indicative of posterolateral subepicardial injury while the orientation of the T sÊ loop almost directly anteriorly is compatible with posterior ischemia.

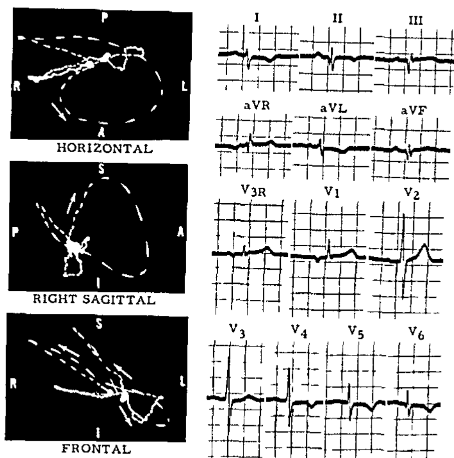


Fig 195 —Electrocardiographic and vectorcardiographic findings in diaphragmatic posterolateral myocardial infarction of uncertain duration

The diagnosis of diaphragmatic infarction is based on the presence of the deep relatively narrow Q waves in leads II III and aVF of the electrocardiogram while the low R wave and embryonic notched S wave in lead V_1 constitutes

... loop is initially inscribed far to the right being situated at +120 in the horizontal plane −50 in the sagittal and −130 in the frontal plane the maximal mean instantaneous QRS vector corresponding to the long axis of the planar QRS loop lies at −170 in the horizontal plane at −40 in the sagittal plane and at −150 in the frontal plane These findings are indicative of diaphragmatic posterolateral infarction One should note that the terminal portion of the QRS sÊ loop in the above figure as well as in the vectorcardiogram in Figure 193 is located to the right posterio ... r is directed to the right slightly ... The T sÊ loop is oriented to the ... ads I and V_6

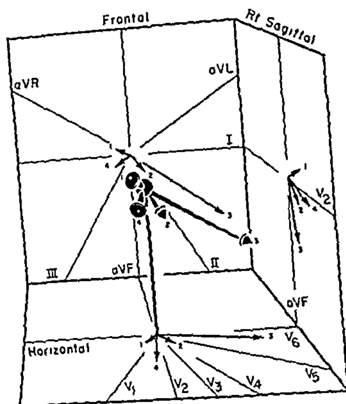
STRICTLY POSTERIOR INFARCTION

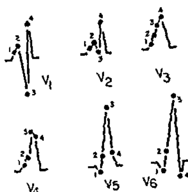
In conventional electrocardiographic nomenclature the term *posterior myocardial infarction* has been applied to infarctions producing diagnostic abnormalities in leads II III and aVF with or without associated reciprocal changes in the anterior precordial leads V_1 to V_3 However in this text the term posterior is used to designate infarctions which give rise to unbalanced forces directed almost straight anteriorly In other words a strictly posterior infarction is one whose effective electrical site is located posteriorly not inferiorly in the left ventricular free wall Until recently strictly posterior infarction was not recognized since this type of infarction does not produce abnormal Q waves in any of the twelve electrocardiographic leads routinely recorded In contrast with the electrocardiogram the vectorcardiogram is frequently diagnostic in strictly posterior infarction The fact must be emphasized however that the ex

... following discussion of strictly posterior infarction is



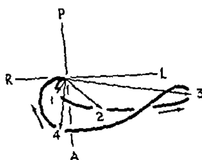
A Strictly Posterior Myocardial Infarction



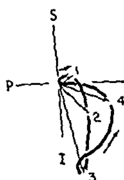


C QRS Deflections Projected on Scalar Leads

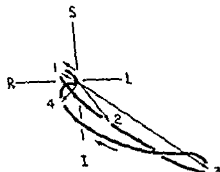
B Instantaneous VA Vectors in Strictly Posterior Myocardial Infarction



Horizontal



Right Sagittal



Frontal

D Planar QRS Loops in Strictly Posterior Myocardial Infarction

(D) show anterior displacement of their afferent or returning *limbs* while the frontal QRS loop is normal

based primarily on earlier observations of Grishman and his investigative group supplemented by our observations in our own series of patients with this type of infarction

The manner in which the diagnostic QRS abnormalities are produced by strictly posterior infarction is not known However strictly posterior infarction behaves electrically as if it were situated in some region of posterobasal left ventricular wall which is activated relatively late in the QRS interval Thus the most marked changes due to the infarction are relegated for the most part to the second half of the QRS deflection and QRS sÊ loop

The Instantaneous VA Vectors

The electrical forces produced in strictly posterior infarction can be presented in simplified manner in terms of the instantaneous VA vectors (see also Fig 196)

0 01 TO 0 04 SECOND VA VECTORS

The 0 01 and 0 02 second VA vectors have essentially normal characteristics since these vectors represent depolarization potentials arising in uninvolved regions of the septum and left ventricle which are activated early in the QRS interval On the other hand the 0 04 second VA vector is more variable in its orientation in that it is situated to the left and either posteriorly or as is more commonly the case anteriorly

Lead V_1 –The 0 01 to 0 04 second VA vectors project on this lead an initial small R wave followed by the downstroke of an S wave which is perhaps somewhat shallower than normal or the initial R wave may be followed by an incisura not reaching the base line or by the slurred upstroke of a secondary R wave The factor determining which of the preceding deflections is recorded is the degree of anterior displacement of the 0 04 second VA vector

Leads I aVL V_5 and V_6 –The 0 01 to 0 04 second VA vectors produce small normal Q waves followed by relatively normal appearing R waves in these leads

0 06 AND 0 08 SECOND VA VECTORS

Following the appearance of the 0 04 second VA vector subsequent instantaneous vectors in strictly posterior infarction tend to develop progressively to the right and anteriorly Thus the 0 06 second VA vector usually extends more anteriorly than to the right while the 0 08 second VA vector projects more to the right than anteriorly (and not infrequently is situated somewhat posteriorly) Whether the anterior displacement of these vectors reflects simply the effect of an anteriorly directed infarction vector on the balance of cardiac forces existing during the second half of the QRS interval or whether an additional mechanism is involved(as for example conduction delay in some portion of the intraventricular conduction system consequent to the infarction) is not known at present

Lead V_1 –Typically the 0 06 and 0 08 second VA vectors project on lead V_1 a relatively tall R wave which may or may not be followed by a small terminal S wave depending on whether the 0 08 second VA vector is located anterior or posterior to the –150 axis of the horizontal reference frame In summary in strictly posterior infarction lead V_1 typically records an RSR deflection a notched or slurred R wave (of lower amplitude than that occurring in right ventricular hypertrophy) or an RS deflection with an R/S amplitude ratio exceeding 1

Leads I aVL V_5 and V_6 –These leads complete the inscription of an R wave followed by a terminal S wave Thus the leads record qRs or qRS deflections in strictly posterior infarction Unless there is concomitant diaphragmatic infarction leads II III and aVF display normal QRS complexes

Vectorcardiographic Findings

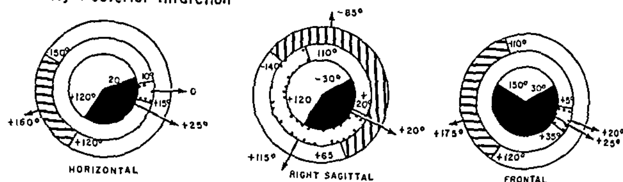
The average orientation and the extreme range of orientations of the mean 0 02 second maximal mean and mean 0 06 second instantaneous QRS vectors of the horizontal sagittal and frontal loops in strictly posterior myocardial infarction with and without diaphragmatic infarction are shown in Table 27 and Figure 197

HORIZONTAL QRS LOOP –The QRS sÊ loop in the horizontal projection is initially written to the right and anteriorly just as normally and then the efferent limb is inscribed to the left and anteriorly After reaching its maximal leftward extent the loop turns in a clockwise direction and moves medially toward the right In strictly posterior infarction the afferent limb is displaced anteriorly and may lie anterior to the efferent limb throughout its entire course In the latter situation the horizontal QRS loop has a clockwise direction of inscription and the terminal portion of the loop is situated to the right and anteriorly or occasionally posteriorly In some cases only the first portion of the afferent limb lies anterior to the efferent limb and in this event the crossing of the afferent

TABLE 27.—ORIENTATION OF THE MEAN 0.02 SECOND, MAXIMAL MEAN AND MEAN 0.06 SECOND INSTANTANEOUS VECTORS OF THE QRS sÊ LOOP IN STRICTLY POSTERIOR MYOCARDIAL INFARCTION WITH AND WITHOUT DIAPHRAGMATIC INFARCTION

	Horizontal			Right Sagittal			Frontal		
	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range	Extreme Range	Av	Usual Range
Strictly Posterior Infarction without Diaphragmatic Infarction									
Mean 0.02 second instantaneous QRS vector	–20 to +120	+25	–20 to +20	–30 to +120	+20	–20 to +100	–30 to –150	+25	–30 to +35
Maximal mean instantaneous QRS vector	–10 to +15	0		+20 to –110	+115	+60 to –140	+5 to +35	+20	
Mean 0.06-second instantaneous QRS vector	+120 to –150	+160	+135 to –175	140 to +65	65	–120 to +15	+120 to –110	+175	+150 to –115
Strictly Posterior Infarction with Diaphragmatic Infarction									
Mean 0.02 second instantaneous QRS vector	–25 to +120	+60	+50 to +120	110 to –40	–65	–60 to –40	–70 to –50	–60	
Maximal mean instantaneous QRS vector	+10 to +25	+20		0 to +70	+35		0 to +45	+25	
Mean 0.06 second instantaneous QRS vector	+50 to –110	160	–175 to –110	–30 to –175	+80	+50 to –175	0 to +120	+55	+45 to +120

Usual Range = range in 85% of cases.



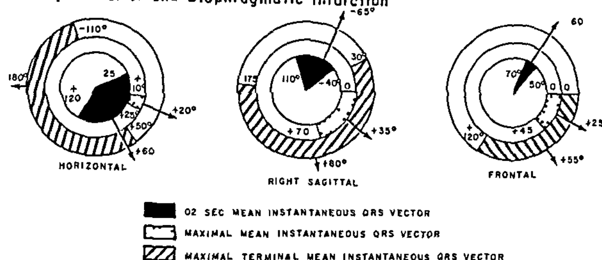


Fig 197 —Range of variation and average orientation of the mean 0 02 second the maximal mean and the maximal terminal mean instantaneous QRS vectors *in strictly* posterior and diaphragmatic–strictly posterior myocardial in farctions

and efferent limbs of the horizontal QRS loop gives the latter a figure of eight configuration the proximal loop of the eight being counterclockwisely inscribed and the distal loop clockwisely inscribed Not uncommonly loops of this configuration display a terminal deflection to the right and far posteriorly In our experience the mean 0 02 second instantaneous vector of the horizontal QRS loop in strictly posterior infarction was found to be oriented just as normally while the maximal mean instantaneous QRS vector tended to shift abnormally far anteriorly As a general rule the mean 0 06 second instantaneous QRS vector in the horizontal projection is located abnormally far to the right and anteriorly which is in striking contrast with the almost directly posterior orientation of the normal mean 0 06 second vector

Right sagittal QRS loop —In strictly posterior infarction the sagittal QRS loop confirms the anterior displacement of the long axis or maximal mean instantaneous vector of the QRS sÊ loop already described in the horizontal projection In general the sagittal loop presents either of the following configurations (1) When the entire afferent limb of the horizontal QRS loop lies anterior to the efferent limb the sagittal loop is written entirely in a counterclockwise direction (2) When only a part of the afferent limb is situated anterior to the efferent limb in the horizontal projection the same condition holds true in the sagittal projection Thus the sagittal QRS loop will have a figure of eight configuration in which the proximal component of the loop is clockwisely inscribed and the distal component counterclockwisely inscribed The mean 0 02 second instantaneous QRS vector in the sagittal projection like that in the horizontal does not differ from the normal in orientation unless there is associated diaphragmatic infarction The maximal mean instantaneous QRS vector (long axis of the sagittal QRS loop) tends to be located somewhat more posteriorly than normal while the mean 0 06 second instantaneous QRS vector is quite

distinctive in that it is directed anteriorly instead of posteriorly as is normally the case

Frontal QRS loop —In the absence of combined diaphragmatic infarction the frontal QRS loop in strictly posterior infarction does not show diagnostic abnormalities since the unbalanced forces produced by this type of infarction are oriented primarily perpendicular to the frontal plane

The T sE loop and the S–T vector in strictly posterior infarction are dealt with at the end of this chapter

The ECG Criteria for Diagnosis

QRS Abnormalities

(Although strictly posterior infarction fails to produce abnormal Q waves in any of the twelve routine electrocardiographic leads QRS changes do appear typically in one or more of the right precordial leads (Needless to say specific criteria for the electrocardiographic diagnosis of strictly posterior infarction have not been formulated as yet) The electrocardiograms from patients with vectorcardiographically diagnosed old strictly posterior infarction which we reviewed displayed the following features

Leads I aVL and V_6 —Abnormal Q waves absent in all cases

Leads V_{3R} *and* V_1 —RSR configuration (rSr' rSR or rsR) in 40% of the cases notched or slurred R or Rs with R/S ratio ≧ 1 in 35% of the cases and QRS configuration of the rS type in lead V_1 in 25% of the cases

It is evident from the description of the electrocardiogram and vectorcardiogram in strictly posterior infarction that the electrocardiographic QRS residuals

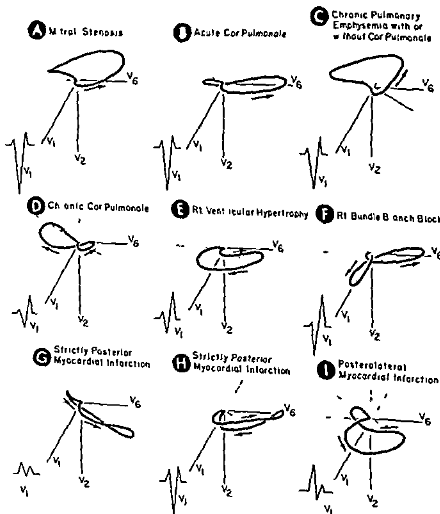


Fig 198 —Horizontal QRS loop patterns which may be accompanied by RSR deflections in lead V_1 of the electrocardiogram The schematic QRS loops in G and H represent two types of loop configurations observed in strictly posterior infarction In all examples of QRS loop configuration except that in F the QRS duration in the electrocardiogram is 0 10 second or less

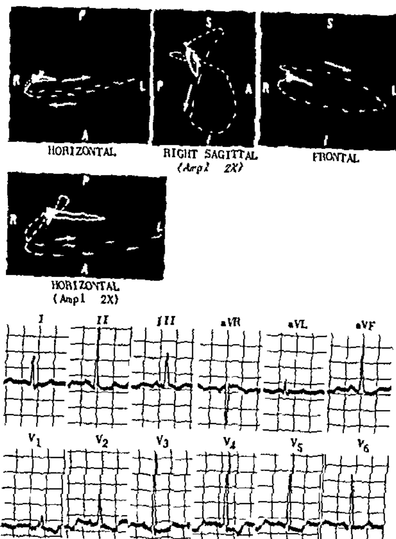


Fig 199 —Electrocardiographic and vectorcardiographic findings in an old or healed strictly posterior infarction One year before these recordings were taken the patient was hospitalized for 6 weeks with a clinically typical picture of acute myocardial infarction (The only finding in the above electrocardio- [illegible] of the figure-of-eight sagittal loop

of this type of infarction can simulate incomplete right bundle branch block right ventricular hypertrophy the pattern of "counterclockwise rotation" or the juvenile precordial lead pattern in which the R waves in right precordial leads are relatively large Not only is the vectorcardiogram useful in differentiating these and other conditions producing RSR deflections in lead V_1 but—of equal importance—it is free from the several handicaps which limit the value of the electrocardiogram in diagnosing strictly posterior infarction (Fig 198) These electrocardiographic limitations are as follows (a) The salient diagnostic feature of infarction an abnormal Q wave or QS deflection appears in none of the routine electrocardiographic leads (b) More often than not the increased amplitude of the R waves in right and midprecordial leads is not particularly striking (c) Although esophageal leads or leads from the posterior thorax may occasionally show diagnostic changes the recording of such leads is a time consuming procedure and the results obtained are often quite difficult to interpret

S–T Vector and Ventricular Repolarization

The authors of this text have rarely had the opportunity to study vectorcardiographically or electrocardiographically strictly posterior infarction in the acute stage However we might predict the abnormalities which would be present in an acute posterior infarction

1 The terminus of the QRS sE loop would be displaced posteriorly and so the right and midprecordial leads of the electrocardiogram would register depressed S–T segments
2 The T sE loop would probably be situated anteriorly and somewhat to the left or right Thus the right and midprecordial leads would display upright T waves during the stage of posterior transmural ischemia

The vectorcardiographic and electrocardiographic findings in old or healed strictly posterior infarction are well exemplified in Figures 199 and 200

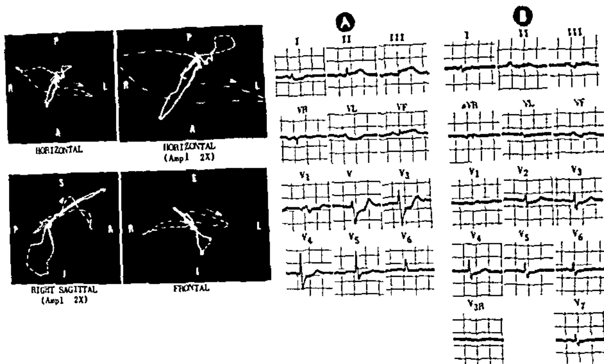


Fig 200 —Electrocardiographic and vectorcardiographic findings in acute diaphragmatic–strictly posterior myocardial infarction

isoelectric level.

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s are clearly di

agnostic of acute diaphragmatic–strictly posterior infarction The large inferiorly oriented loop in the right sagittal projection is the P loop whose size merely reflects the extreme diminution in QRS forces apparently consequent to the infarction.

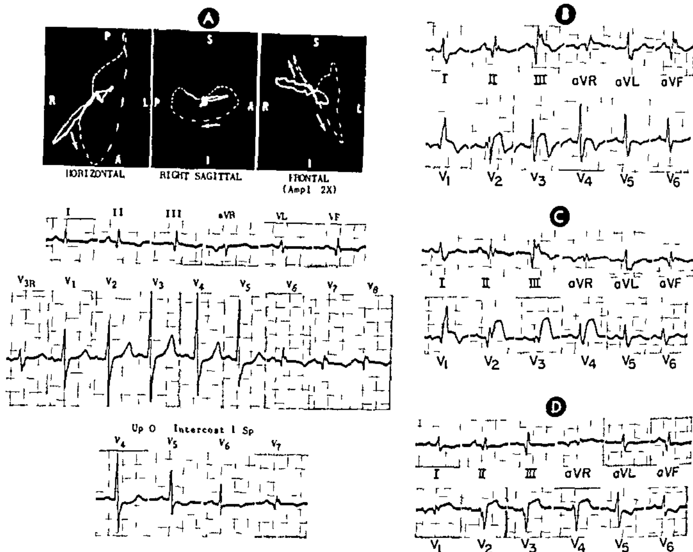


Fig 201 —Electrocardiograms and vectorcardiogram in combined diaphragmatic posterolateral and subsequent anter … ocardial infarctions

… infarction The diagnostic QRS abn … ugh V and in V … V and in V up one intercostal space In addition the S–T segments … through V_4 up one intercostal space while leads *I II III aVF V through V and V through V_7 up one intercostal space all register* inverted T waves These findings are compatible with diaphragmatic posterolateral infarction in the recent past Corresponding *abnormalities in the vectorcardiogram are as follows the horizontal QRS loop is written abnormally far ante*riorly over one half of the loop being situated anterior to the frontal plane … written … riorly and slightly to the right the QRS sE loop doe … and … in t … of t …

hos … terminal S waves have appeared … are recorded in leads aVR and V_1 … vated S–T segments and inverted T waves The abnormalities previously described as being diagno… *matic infarction persist in this record but the signs of posterolateral infarction have disappeared* The record can therefore be interpreted as showing an acute anterior myocardial infarction an old diaphragmatic myocardial infarction and right bundle branch block

C … have appeared in leads V and V confirming the initial im*pressi*…

D … 0 10 … with second …

Miscellaneous Cardiac Abnormalities

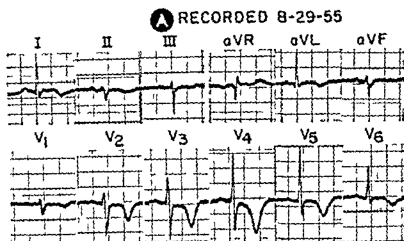
INFARCTIONS IN COMBINED LOCATIONS

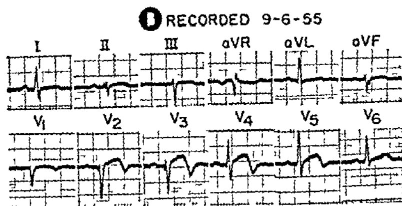
Myocardial infarction frequently involves several different areas of the left ventricle concomitantly or may occur with an older infarction of a different location. As a general rule, the infarction vector in combined infarction is the resultant of the component infarction vectors of each aspect of the ventricular wall involved. For example, in diaphragmatic anterolateral infarction the infarction vector points superiorly to the right and posteriorly; in diaphragmatic posterolateral infarction, it is directed superiorly to the right and anteriorly; and in diaphragmatic anteroseptal infarction, it points to the left, posteriorly, and superiorly. In most instances, each of the components of a combined infarction produces changes individually recognizable in the electrocardiogram and vectorcardiogram, so that infarction in one region of the left ventricle ordinarily does not interfere with the diagnostic changes produced by infarction of another region. Perhaps the major exception to this rule—but a rare one—is combined infarction of anterior and posterior walls of the left ventricle (Figs. 201 and 202). In a case of infarction of this type, the authors of this text were able to follow serial electrocardiograms recorded during the evolution of the two infarction patterns. The initial electrocardiograms showed the evolution of typical QRS complex, S–T segment, and T wave abnormalities of anterior infarction. A short time later, with a sudden deterioration in the patient's clinical status, the electrocardiographic precordial leads, which previously had displayed QS deflections or abnormally low R waves, showed R waves of relatively normal size and a normal precordial QRS transi

notching on the down-stroke of the S wa es nd T

II III

iyocar





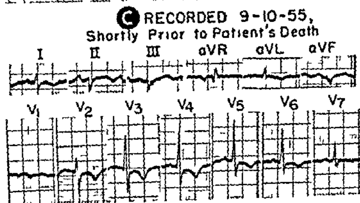


Fig 202 —Electrocardiographic findings in a man 53 with acute posterior myocardial infarction superimposed on recent anterior myocardial infarction He was admitted to the hospital on August 19 1955 after 5 hours of severe chest pain His clinical ~ s stormy and ¹

ı ... ay in
\ ... s in leads V_3 and V_4 compatible with anterior myocardial ischemia On September 6 (record B) the following changes were noted QS deflections in lead V_1 small initial Q waves with abnormally low R waves in leads V_2 and V_3 elevated S–T segments in leads V_2 through V_5 and inverted T waves in leads V through V_6—changes considered compatible with acute anterior or anteroseptal myocardial infarction On September 10 he developed more severe chest pain and pulmonary edema and expired. Record C obtained shortly before his death showed a vibratory downward ventricular beat of low voltage in lead V_1 on the other hand the R waves in leads V_2 through V were if anything larger than those in A The S–T segments in these same leads were slightly depressed On the basis of the trend toward "normalization of the QRS deflections present in C as compared to B record C was interpreted as suggestive of superimposed acute posterior myocardial infarction on an antecedent anteroseptal infarction Postmortem examination disclosed a healing infarction of the apex and anterior wall of the left ventricle with mural thrombus and recent infarction of what would correspond in terms of electrical location to the posterior wall and septum of the left ventricle with terminal rupture of the posterior ventricular wall Occlusions of the anterior descending branch of the left coronary artery and of the circumflex branch of the left coronary artery were noted In summary the anteroseptal infarction lessened or abolished anteriorly directed instantaneous QRS forces causing the balance of forces to shift posteriorly and producing Q waves and low R waves in the anterior precordial leads Subsequent posterior infarction removed posteriorly directed instantaneous forces so that the balance of electrical forces returned more nearly to the normal and significant R waves reappeared in the anterior precordial leads as the only evidence of the later infarction The ... could
S–T segment depre ... eleva
... ı pos-
ı
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tion. In addition there was slight S–T segment depression in the precordial leads and the T waves previously inverted had either become low upright or less deeply inverted The electrocardiographic diagnosis of acute strictly posterior myocardial infarction superimposed on recent strictly anterior infarction was confirmed at postmortem If the patient had lived electrocardiograms recorded at some future date conceivably might have shown no evidence of either infarction since the electrical effects of one would tend to counterbalance the effects of the other That such a course of events can occur (as illustrated in Figures 201 and 202) constitutes clinical evidence of the validity of the dipole or vector theory

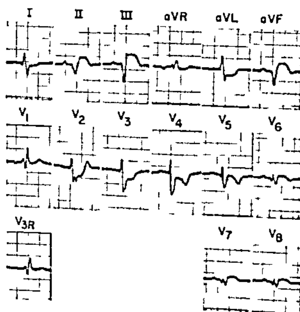
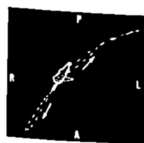


Fig 203 –Electrocardiographic findings in right bundle branch block with a superimposed acute diaphragmatic posterolateral myocardial infarction The presence of right bundle branch block is indicated by the prolonged QRS interval the wide terminal R waves in leads V and V and the wide terminal S waves in leads I and V through V_6. The QS deflec

in leads V through V are equally diagnostic of acute posterolateral infarction. The depressed S–T segments in leads V and V are probably related in a reciprocal manner to posterior subepicardial injury







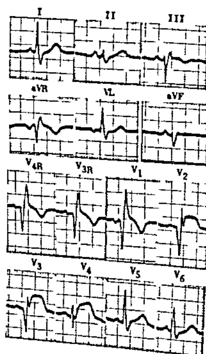
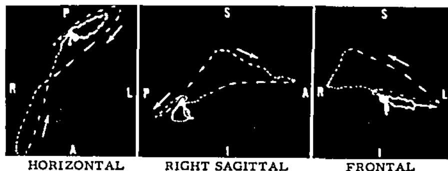


Fig 204 – Electrocardiographic and vectorcardiographic findings in right bundle branch block with superimposed acute anteroseptal myocardial infarction The vectorcardiographic QRS sÊ loop is written initially to the left posteriorly and inferiorly indicating anteroseptal infarction while the terminal portion of the QRS sÊ loop is slowly inscribed anteriorly to the right and slightly superiorly signifying right bundle branch block Although an anteriorly directed S–T vector is present in the vectorcardiogram, its magnitude is smaller than one might have expected in view of the marked S–T segment elevation in leads V_4 through V of the electrocardiogram The reason for this discrepancy is that the vectorcardiogram was recorded several days later than the electrocardiogram when the subepicardial injury was subsiding

MYOCARDIAL INFARCTION WITH BUNDLE BRANCH BLOCK

The single fact most important to understanding the electrocardiographic and vectorcardiographic abnormalities in coexisting myocardial infarction and bundle branch block is that infarction of the septum or left ventricular free wall manifests itself electrically, as a general rule, only at and during the time the involved myocardium would normally undergo activation. Since normally septal activation occurs during the first 0.02 second of the QRS interval and activation of all but the basal portions of left ventricular free wall takes place between 0.02 and 0.04 second of the QRS interval, anteroseptal infarction, for example, alters the first 0.02 second of the QRS complex, while left ventricular free wall infarction (involving, as it frequently does, a portion of the septum as well) produces abnormalities of the first 0.04 second of the QRS deflection in the form of abnormal Q waves or changes in the size of the R waves. However, *if onset of left ventricular activation* is delayed and septal activation altered as the result of an intraventricular conduction defect, the electrical effect of a superimposed infarction is modified. In the case of left ventricular free wall infarctions, the effect of the infarction is shifted to a later part of the QRS complex and appears coincident with the delayed onset of free wall activation whenever that occurs in the QRS interval. If activation of the left ventricle not only is delayed in onset but takes place in an abnormal or aberrant manner, then the QRS abnormalities are both late in onset and modified in appearance in comparison with the QRS changes produced by the same type of infarction during normal intraventricular conduction. Inasmuch as most clinical infarctions involve interventricular septum and/or free wall of the left ventricle, the crucial factors determining the effect of coexisting bundle



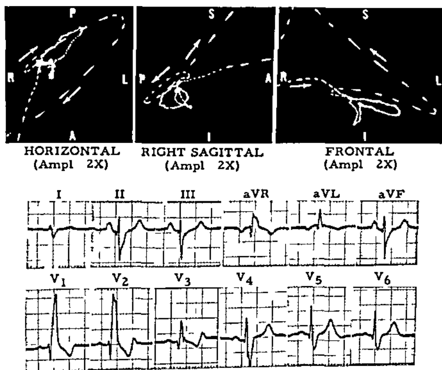


Fig 205 — Electrocardiographic and vectorcardiographic findings in right bundle branch block and coexisting old anterior myocardial infarction.

The sole electrocardiographic finding suggestive of an old anterior infarction is the low initial R wave in lead V_1.

In the vectorcardiogram, however, the horizontal QRS loop shows clockwise inscription of both its initial rightward and anterior deflection and subsequent leftward and posterior portion, both findings probably being related to posterior displacement of the efferent limb of the loop. The corresponding components of the sagittal QRS loop also exhibit a reversed direction of inscription. These early abnormalities of the QRS sÊ loop are diagnostic of anterior infarction. The large rightward and anterior terminal deflection of the horizontal QRS loop and the superior orientation of both sagittal and frontal QRS loops are typical of the variant right bundle branch block pattern described in Chapter 17.

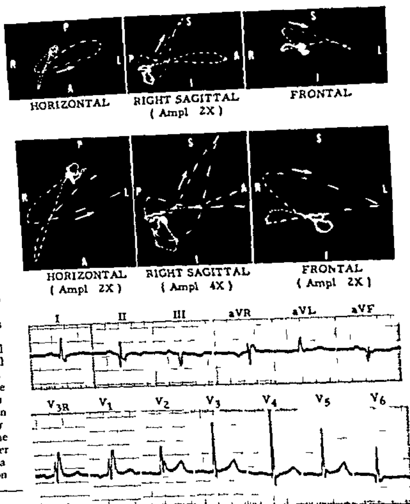


Fig 206 — Electrocardiographic and vectorcardiographic findings in right bundle branch block with coexisting old diaphragmatic posterolateral myocardial infarction

In the electrocardiogram the deep but slender Q waves in leads II and aVF and the QS deflection in lead III are compatible with diaphragmatic infarction The presence of posterolateral involvement might be suspected from the prominent initial R waves and shallow S waves in leads V and V_1 and the somewhat prominent Q wave in lead V

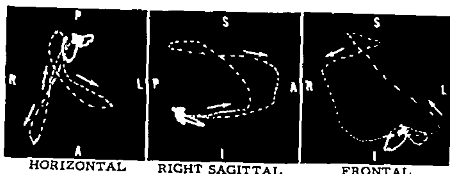
The vectorcardiographic abnormalities diagnostic of diaphragmatic posterolateral infarction and right bundle branch block are obvious There is a large initial deflection of the QRS sE loop abnormally far to the right anteriorly and superiorly The mean 0 02 second instantaneous QRS vector lies at +135 in the horizontal plane −50 in the sagittal plane and −150 in the frontal plane As will be recalled the 0 02 second vector does not normally lie to the right of +100 in the horizontal plane or superior to −40 in the sagittal plane and so the abnormal orientation of this vector in the above vectorcardiogram is further confirmation of the presence of diaphragmatic posterolateral infarction

branch block on the changes due to infarction are therefore dependent on the time and manner in which the septum and left ventricle are activated.

Right Bundle Branch Block

In right bundle branch block left ventricular activation takes place just as it does when intraventricular conduction is normal Accordingly the presence of right bundle branch block in no way obscures the abnormal initial and/or early QRS forces characteristic of myocardial infarction In turn the electrocardiographic and vectorcardiographic features diagnostic of right bundle branch block which it will be remembered alters the terminal mean instantaneous QRS vectors are not disturbed by superimposed infarction Thus coexisting right bundle branch block and myocardial infarction are individually recognizable in the vectorcardiogram and electrocardiogram because the electrical effects of the two conditions appear at different times in the QRS interval One possible exception to this rule is right bundle branch block with strictly posterior myocardial infarction As will be recalled this type of infarction typically affects the later instantaneous QRS vectors causing them to be displaced anteriorly Another common although by no means invariable abnormality in this type of infarction is anterior displacement of the long axis of the QRS sE loop The only cases of strictly posterior infarction occurring with right bundle branch block which the authors of this text have been able to recognize with any assurance in the vector cardiogram are those in which the long axis of the QRS sE loop was shifted farther anteriorly than is usually observed in uncomplicated right bundle branch block In none of these cases was it possible to make the electocardiographic diagnosis of strictly posterior infarction with any degree of certainty

In the acute stage of infarction superimposed on right bundle branch block the secondary S–T seg



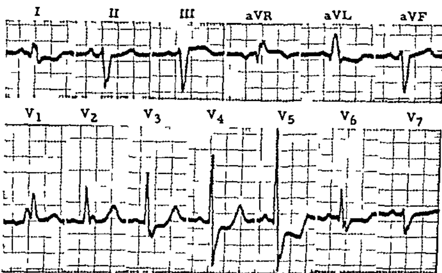


Fig 207 — Electrocardiographic and vectorcardiographic findings in right bundle branch block with co-existing old posterolateral myocardial infarction

The small Q waves in leads I V_6 and V_7 in the electrocardiogram can not be considered diagnostic of infarction The broad initial R of the RR deflection in lead V_1 is perhaps the best electrocardiographic evidence of posterolateral infarction but even this finding is somewhat equivocal

The QRS sE loop of the vector ing abnormalities diagnostic of posterolateral infarction There is marked anterior displacement of both efferent and afferent limbs of the horizontal and sagittal QRS loops causing a complete reversal in the direction of inscription of the first half of the horizontal loop The frontal QRS loop is inscribed a short distance to the left and then turns abruptly superiorly and medially reflecting medial displacement of the greater part of the efferent limb of the loop An S–T vector directed to the right and inferiorly is probably representative of digitalis effect or lateral subendocardial injury The T sE loop is situated anteriorly and slightly inferiorly indicating posterior ischemia

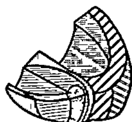
ment and T wave changes of the right bundle branch block modify to some extent but rarely obscure the S–T segment and T wave abnormalities characteristic of acute infarction

In the cases of right bundle branch block with myocardial infarction studied by Dodge and Grant the effective electrical location of the infarction was found to be diaphragmatic in almost 50% of the electrocardiograms As Dodge and Grant pointed out the right ventricle and a major portion of the right intraventricular conducting system lie within the distribution of the right coronary artery and occlusion of this vessel is responsible for most cases of diaphragmatic myocardial infarction Dodge and Grant found that the site of infarction in the remaining half of the patients (with right bundle branch block) was divided about equally between the several types of anterior infarction and posterior infarction (Figs 203–207) Grant and Dodge also studied a series of cases with left bundle branch block in which electrocardiograms recorded before onset of the conduction disturbance showed myocardial infarction Interestingly enough over one half of the electrocardiograms showed diaphragmatic infarction and the distribution of the site of infarction in the remaining differed from that in right bundle branch block only in that strictly anterior infarction was present less commonly than either anterolateral or posterior (strictly posterior and posterolateral) infarction On the other hand in the cases in which the electrocardiogram resembled left bundle branch block but in reality represented peri infarction block anterolateral infarction was the type of infarction most frequently present in tracings recorded before onset of intraventricular block The explanation of Grant and his associates for this finding is given on page 337

Left Bundle Branch Block

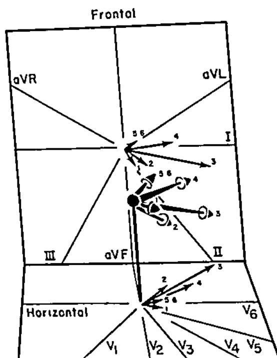
Unlike right bundle branch block left bundle b... ...le it quite difficult often impossible ... of the ... t... ... diagnostic problem is somewhat less ... S-T segment and T wave changes due to infarction can sometimes be detected despite the presence of

... ...nch block ... tri... or ... second... sible for ...



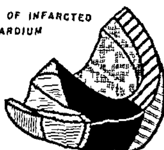
1. INITIAL ACTIVATION OF APICO ANTERIOR RIGHT VENTRICULAR WALL
2. RIGHT-TO-LEFT SEPTAL ACTIVATION AND ACTIVATION OF RIGHT VENTRICULAR FREE WALL
3. COMPLETION OF SEPTAL AND RIGHT VENTRICULAR ACTIVATION
4. INITIAL ABERRANT ACTIVATION OF BASAL LEFT VENTRICULAR WALL
5. ACTIVATION OF POSTERIOR LATERAL AND ANTERIOR LEFT VENTRICULAR WALL
6. COMPLETION OF ACTIVATION OF ANTERIOR WALL OF LEFT VENTRICLE

A Sequence of Activation of Septum and Ventricles

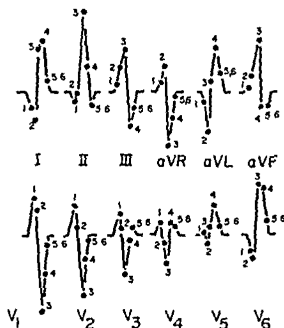


B Instantaneous VA Vectors in Uncomplicated Left Bundle Branch Block

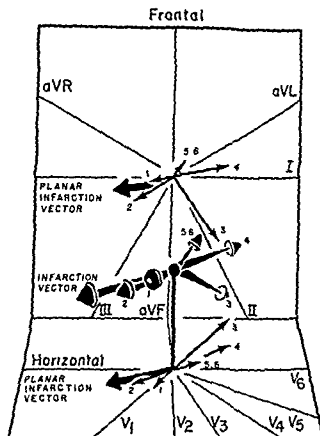
Fig 208 —Instantaneous VA vectors in uncomplicated left bundle branch block. The septal ventricular activation sequence the VA vectors themselves the QRS deflections projected on the scalar leads of the electrocardiogram and the corresponding planar QRS loops were described in Figure 141 which is reproduced here (in part) mainly to serve as a basis for comparison with Figures 209 and 210



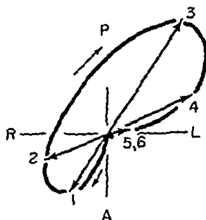
A Schematic Representation of a Septal Infarct

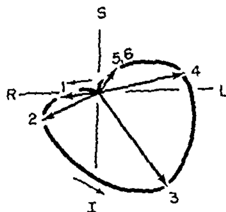


C QRS Deflections Projected on Scalar Leads



B Instantaneous VA Vectors in LBBB Complicated by Extensive Septal Infarction



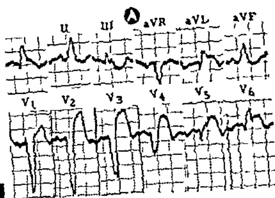


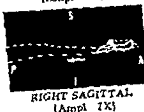
D Planar QRS Loops in Left Bundle Branch Block Complicated by Extensive Septal Infarction

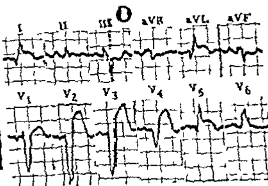
Fig 209 —(*Legend on facing page*)











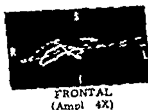


Fig 210 —Electrocardiographic and vectorcardiographic findings in left bundle branch block complicated by acute

s

l

Electrocardiogram B obtained from the same patient several days later shows minor ...

wave in lead V_1 is taller than the R waves in leads V through V In B the corresponding abnormality in the planar QRS loops consists of a large anterior rightward and inferior early deflection of the horizontal and frontal QRS loops

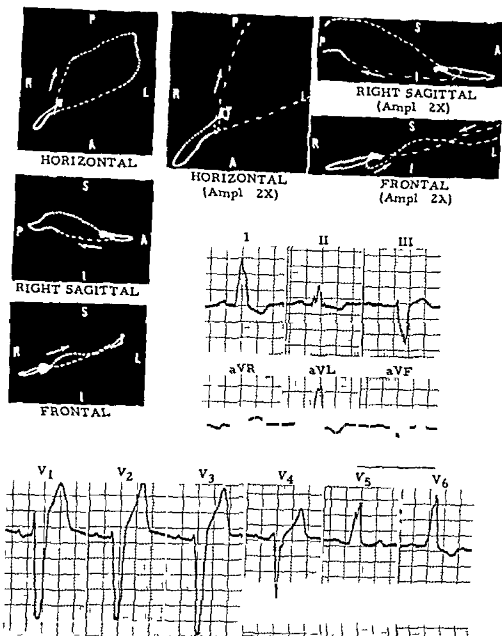


Fig 211 —Electrocardiographic and vectorcardiographic findings in left bundle branch block with coexisting old septal infarction

Although in the electrocardiogram minute Q v … more suggestive of septal infarction is the relatively tall R wave in le… through V

In the vectorcardiogram the QRS sÊ loop is ch… there is an early deflection of the loop to the right anteriorly and inferiorly indicative of septal infarction

in left bundle branch block they are as follows (Fig 208)

1 Septal depolarization from beginning to end occurs in a right to left direction and (at least in dogs with surgically produced left bundle branch block) requires about 0 04 second for completion As a result the instantaneous QRS vectors during the first 0 02 second of the QRS interval are oriented toward the left ventricle rather than away from it while the instantaneous vectors appearing during the next 0 02 second are also directed to the left and posteriorly whether the left ventricular free wall happens to be infarcted or not Thus in left bundle branch block leads I and V_6 record positive voltages during the first 0 04 second of the QRS interval regardless of the presence or absence of a free wall infarction

2 Since activation of left ventricular free wall does not commence in left bundle branch block until the second half of the QRS interval the electrical effects of a free wall infarction likewise do not appear until

this portion of the QRS interval Thus free wall infarctions occurring in left bundle branch block are electrocardiographically *"silent" during* the first half of the QRS interval on the other hand it is only during this period that evidence of septal infarction *complicating left bundle* branch block is present in the electrocardiogram

From the standpoint of their *recognition, infarctions complicating left bundle branch block can* therefore be separated into *two* general categories (*a*) septal infarctions which alter the first 0 04 second of *the QRS deflection and* (*b*) *left ventricular* free wall infarctions which affect the final 0 04–0 06 second of the QRS deflection

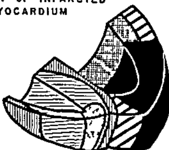
Massive infarction of the interventricular septum –An extensive *infarction of the septum usually* abolishes the instantaneous electrical forces caused by right to-left septal depolarization thereby allowing electrical forces (presumably arising in the right ventricle) *to dominate temporarily the electrical field of the heart* (Fig 209) Consequently the initial and early leftward directed instantaneous vectors characteristic of left bundle branch block are rotated to the right, away from the left ventricle and project Q

[illegible]

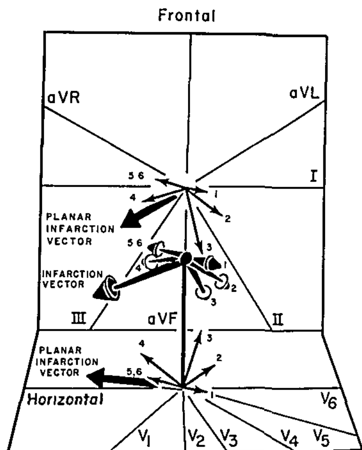
this lead and diminishes *in size from* right *to* left across *most* of the precordium The reversal in the R wave transition is related to the fact that as the result of the infarction the instantaneous vectors of the first 0 04 second of the QRS interval are directed to the right while subsequent instantaneous vectors develop in a clockwise direction to the left and posteriorly in *the* horizontal plane—that is away from the other precordial leads When finally after considerable delay left ventricular activation commences the instantaneous *vectors* appearing after 0 04 second are oriented toward the left ventricle just as is the case in uncomplicated left bundle branch block and these vectors project *terminal* R waves on leads I aVL and V_6 and a terminal S wave on lead V_1 (Figs 210 and 211)

Infarction of left ventricular free wall –Since the late instantaneous QRS vectors in left bundle branch block are determined by delayed left ventricular activation it is these instantaneous *vectors* which are altered by infarction of *the left* ventricular free wall (Fig 212) The failure of the infarcted portion of the free wall *to* generate QRS potentials during the terminal 0 04–0 06 second of the QRS *interval* allows oppositely directed forces to become preponderant so that the late *instantaneous vectors* are displaced away from the effective electrical site of the infarction As it so happens the displacement of the terminal vectors *can ordinarily* be recognized only if the *vectors project* negativity on a lead which in left bundle branch block would otherwise be expected to register terminal positivity (or the converse—*that is terminal positivity* on a lead ordinarily recording terminal negativity in left bundle branch block a situation which will be discussed later) For this reason free wall infarction *in left bundle branch block usually can* be detected only if it produces terminal S waves in leads I aVL, V_5 and V_6 Lateral *infarction* is therefore more likely to be recognized than *infarction* of other portions of left ventricular wall since all routine electrocardiographic leads other than those cited above can and usually do record terminal S waves in uncomplicated left bundle branch block *In* lateral *free* wall infarction electrocardiographic leads which prior to infarction had recorded the wide slurred or notched R waves typical of left bundle branch block afterward typically display RS deflections the initial R wave resulting from undisturbed right to-left septal depolarization and the terminal S wave reflecting *the lateral* wall infarction *If there is* septal as well as free wall *infarction* leads I aVL and V_6 may display notched QS deflections or qrS complexes In addition W shaped deflections may appear *in* leads V_4 and V_5

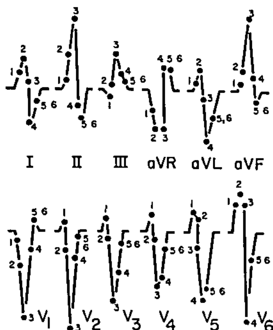
When *lead* V_6 *and/or lead I* record Q waves despite other electrocardiographic findings suggestive of complete left bundle branch block diagnostic precedence *should always be given to the* possibility of septal infarction not only because of the serious implications of the latter but also because of the frequency with which left bundle branch block and infarction occur together clinically However the following facts should be kept in mind in evaluating *the significance of Q* waves *in* leads I aVL and V_6 in the presence of left bundle branch block (1) In uncomplicated left bundle branch block leads I and V_6 *can sometimes display* Q waves but the duration *of the* Q waves does not usually attain 0 02 *second in* the absence of infarction On the other hand it is not unusual for lead aVL *to show deep* wide Q waves in uncomplicated *left bundle* branch block (2) Grant *and others* have pointed out that diffuse *intraventricular* block and peri infarction block neither of which alters the initial QRS *forces* can easily be mistaken for left bundle branch block Since Q waves may be present *in* leads I and V_6 in diffuse intraventricular block (in which there is merely *prolongation* of the QRS interval with little change in QRS configuration) and peri *infarction* block these conditions may



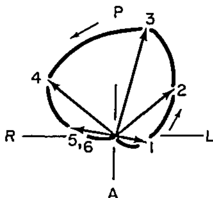
A Schematic Representation of an Anterolateral Myocardial Infarct

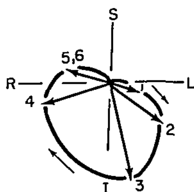


B Instantaneous VA Vectors in LBBB Complicated by Anterolateral Myocardial Infarction



C QRS Deflections Projected on Scalar Leads





D Planar QRS Loops in Left Bundle Branch Block Complicated by Anterolateral Myocardial Infarction

Fig 212 —(*Legend on facing page*)

therefore occasionally simulate left bundle branch block with septal infarction

The presence of terminal S waves in leads I and V_6 in what is otherwise a typical left bundle branch block is of more dubious reliability in establishing the diagnosis of free wall *infarction* unless the change in QRS configuration is observed to develop in serial electrocardiograms recorded during an episode clin ically suspicious of infarction or is accompanied by S–T segment and T wave changes compatible with acute infarction (S–T segment and T wave abnor malities of infarction superimposed on left bundle branch block are discussed at the end of this section)

If the infarction is healed and only one electrocar diogram is available it becomes quite difficult to evaluate the significance of terminal S waves in lead V_6 in particular The reasons for this follow

1
in lea
bundl
on two factors first the frontal QRS forces in left bundle branch block as represented by the frontal QRS loop of the vectorcardiogram tend to be oriented markedly superiorly and to the left and to develop in a counterclockwise direction and second, as pointed out in an earlier chapter the exploring electrode of lead V_6 is more or less routinely applied well below the horizontal plane through the cardiac dipole center Since the indifferent electrode of lead V_6 is at the central terminal or in effect at the electrical cen ter of the heart the low position of the chest electrode causes the lead axis of lead V_6 to be tilted downward This in turn may cause the terminal portion of the frontal QRS loop in left bundle branch block to project on the negative rather than the positive half of the lead axis of lead V_6 To determine whether this factor is responsible for the presence of a terminal S wave in lead V_6 the latter lead can be recorded up one intercostal space

2 Another explanation for the terminal S waves sometimes observed in lead V_6 in uncomplicated left bundle branch block is that there may be a marked shift of the precordial QRS transition to the left in such cases In this event lead V_6 may still lie to the right of the transition point Precordial leads should be recorded farther to the left of lead V_6 to confirm or refute this possibility

3 In our experience the electrocardiographic findings which accompany the vectorcardiographic pattern of so-called left ventricular hypertrophy with t ... on delay" can readily be confused

QRS vectors are usually not disturbed The S wave in lead V_6 which characterizes "left ventricu lar hypertrophy with terminal conduction delay" is produced by a terminal return of the QRS sE loop to the right posteriorly and superiorly Hence the simi larity of the electrocardiographic patterns of the con ductive disturbances

4 Grant has found that in a small percentage of strictly anterior infarctions peri infarction block may lead to QRS interval prolongation and to displacement of the terminal QRS vectors to the right and superiorly When this happens the electrocardiogram tends to resemble left bundle branch block with S waves in lead V_6

S–T segment and T wave abnormalities in diagnosis of infarction with left bundle branch block — Al though an acute myocardial infarction involving the left ventricular free wall in left bundle branch block may or may not produce recognizable changes in the QRS deflection the presence of an infarction may sometimes be suspected from the direction of displacement of the S–T segments Specifically when the S–T segments in a given lead in left bundle branch block are shifted in a direction just the opposite of that anticipated subepicardial injury and hence infarc tion, may be indicated It will be recalled that in uncomplicated left bundle branch block, leads recording upright QRS deflections characteristically display depressed S–T segments and inverted T waves while downwardly directed QRS deflections are typically followed by upright T waves with S–T segment elevation The mechanism of these S–T seg ment and T wave abnormalities (discussed in Chapter

6) consists in brief of reversal in the over-all direction of ventricular repolarization because of the altered depolarization process. Moreover, as was explained in the section dealing with ventricular gradient in Chapter 6, the larger the area of the QRS deflection the greater the displacement of the S–T segment and T wave in the *opposite* direction. In uncomplicated left bundle branch block the consistent relationship existing between the direction and area of the QRS complex and the direction and degree (area enclosed by the S–T segment) of S–T segment deviation and the direction and size of the T wave indicate that the S–T and T abnormalities in left bundle branch block are secondary in type. This being the case, if one were to find that the R waves of large area characteristically recorded in leads V_5 and V_6 in left bundle branch block are followed by elevated S–T segments (or by isoelectric S–T segments if the R wave area was very great) in these leads, one could infer from this that a large lateral subepicardial injury vector must be present to nullify or reverse the expected secondary shift in the S–T segment. This in turn would raise the question of acute infarction. By the same token, S–T segment depression in right precordial leads in left bundle branch block should raise the question of posterolateral subepicardial injury, possibly infarction.

The VCG Findings in Infarction with Left Bundle Branch Block

As yet there have been few reports describing the vectorcardiographic findings in left bundle branch

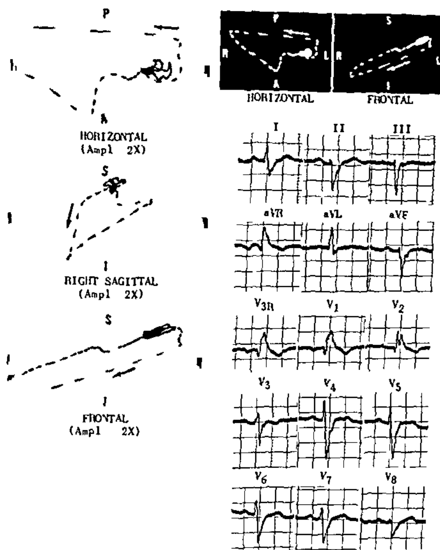


Fig 213 —Electrocardiographic and vectorcardiographic findings in intraventricular block of unknown type

In the electrocardiogram the QRS

slurred terminal R waves are present in leads V[illegible] through V[illegible] and terminal S waves are present in the left precordial leads. The electrocardiogram presents many of the features which the authors of this text consider characteristic of the variant type of right bundle branch block, but the vectorcardiogram does not support this diagnosis.

In the vectorcardiogram the QRS sÊ loop is written initially to the left, slightly anteriorly and slightly superiorly. In the horizontal projection the QRS loop is written in a counterclockwise direction briefly to the left and then the loop turns posteriorly and moves rapidly to the right. The afferent limb of the horizontal QRS loop is then written to the right and slightly anteriorly and shows some evidence of conduction delay. The greater portion of the sagittal QRS loop is written in a counterclockwise direction inferiorly, at first posteriorly and then anteriorly. The configuration of the QRS sÊ loop in each of its planar projections is not that usually observed in right bundle branch block. It is possible that this

tail in the text)

block complicated by myocardial infarction Richman and Wolff described four cases of left bundle branch block

were typical of left bundle branch block; the chest leads were suggestive of right bundle branch block In the vectorcardiograms recorded in these cases the QRS sE loop generally was located to the right, superiorly and either slightly anteriorly or posteriorly The initial vectors of the QRS sE loop in each case were directed anteriorly and inferiorly with or without leftward deviation while the terminal portion of the loop which showed conduction delay returned on the right, superiorly and posteriorly Richman and Wolff ascribed the electrocardiographic and vectorcardiographic abnormalities just cited to left bundle branch block complicated by septal lateral and diaphragmatic myocardial infarction

Occasionally we have observed electrocardiograms and vectorcardiograms which presented some of the features described by Richman and Wolff (Fig 213) However since preinfarction records were not available and postmortem correlation was lacking in our cases we cannot be certain that these cases represented additional examples of left bundle branch block masquerading as right bundle branch block

In the small series of cases having electrocardiograms compatible with the diagnosis of left bundle branch block and septal infarction which we studied vectorcardiographically the principal findings were these (Figs 210–211)

1 There was a prominent initial deflection of the horizontal QRS loop to the right and anteriorly the maximal mean instantaneous vector of this portion of the loop lying between +95 and +145 In uncomplicated left bundle branch block, when an anteriorly directed initial deflection is present the maximal instantaneous vector is rarely situated to the right of +90 and never to the right of +95 and the initial deflection is usually counterclockwise direction In con

plicated by septal infarction generally clockwise direction of inscription
2 The duration of the early portion of the horizontal QRS loop situated to the right of the midline ranged from 0 02 to over 0 03 second in the cases of septal infarction with left bundle branch block However in the few instances of uncomplicated left bundle branch block with a rightward initial deflection of the horizontal QRS loop observed by us the total duration of this part of the loop did not exceed 0 015 second and usually was far less
3 The maximal rightward mean instantaneous vector of the frontal QRS loop ranged in orientation between +130 and −175 while in most cases of uncomplicated left bundle branch block it was impossible to distinguish a rightward initial deflection
4 In one case of acute septal infarction with left bundle branch block the electrocardiogram showed elevated S–T segments and inverted T waves in leads I aVL, V_5 and V_6 and corresponding abnormalities were present in the vector cardiogram The terminus of the QRS sE loop was displaced to the left, anteriorly and slightly inferiorly indicating the presence of a similarly directed S–T vector The T sE loop was oriented to the right anteriorly and inferiorly
5 Except for the findings just cited, the QRS sE loop in left bundle branch block with septal infarction otherwise resembled that in uncomplicated left bundle branch block

INFARCTION OF THE INTERVENTRICULAR SEPTUM

Septal infarction occurs frequently in combination with infarction of left ventricular free wall but rarely if ever is the septum the sole site of involvement In the series of septal infarctions studied electrocardiographically and pathologically by Sodi Pallares and his associates and by Wolff not a single case of septal infarction unaccompanied by free wall involvement was observed, although Myers apparently found several such cases in his series Be that as it may septal infarction is for all intents and purposes almost invariably associated with free wall infarction Septal involvement should be suspected if any of the following abnormalities appear in the electrocardiogram

1 The presence of an extensive infarction of either anterior or inferoposterior walls of the heart
2 The presence of both anterior and inferoposterior infarctions particularly if bundle branch block appears temporarily or permanently at some time during the evolution of the infarction
3 QS deflections in leads V_1 to V_4 or the absence of an R wave in any of leads V_2 V_3 or V_4 if in adjacent leads to the right there is initial positivity

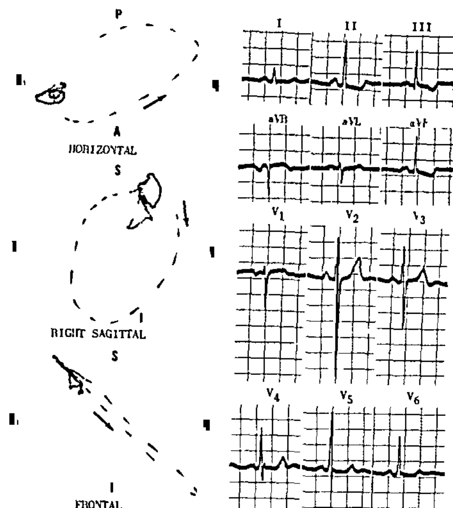


Fig 214 —*Electrocardiogram and vectorcardiogram in septal fibrosis*

Note in the electrocardiogram that normal septal Q waves are absent in leads I aVL V_5 and V_6 This finding in conjunction with the lack of evidence of anteroseptal myocardial infarction is strongly suggestive of fibrosis of the interventricular septum

The corresponding abnormality in the vectorcardiogram consists of the inscription of the initial portion of the QRS s£ loop to the left anteriorly and slightly superiorly There is nothing in the vectorcardiogram to suggest the presence of anteroseptal myocardial infarction or incomplete left bundle branch block The abnormal direction loop is discordant to the long axis of the QRS s£ loop The vectorcardiogram therefore corroborates the electrocardiographic impression of septal fibrosis and also displays a nonspecific T s£ loop abnormality

4 Q waves in leads V_5 and V_6 when there is complete left bundle branch block or in leads V_1 and V when there is complete right bundle branch block Q waves in incomplete bundle branch block do not necessarily signify septal infarction

5 The sudden onset of left or right bundle branch block or of atrioventricular block during an episode clinically compatible with myocardial infarction

FIBROSIS OF THE INTERVENTRICULAR SEPTUM

Healed septal infarction can be simulated pathologically by coalescent foci of fibrosis in the septal muscle The diagnostic electrocardiographic abnormality which Burch has observed in cases of septal fibrosis consists of absence of normal septal Q waves in leads I V_5 and V_6 Normally the small Q waves in these leads reflect initial septal activation in a left to right direction more precisely than does the initial R wave in lead V_1 which also results from depolarization of *paraseptal portions of the left and right* ventricular free walls Consequently the presence or absence of an initial R wave in lead V_1 would *not* be expected to correlate as well with the presence or absence *of septal fibrosis* as the presence or absence *of Q waves in leads I V_5 and V_6 In occa*sional vectorcardiograms we have observed that the horizontal and sagittal QRS loops were written initially to the left anteriorly and inferiorly but were otherwise normal in configuration and orientation (Fig 214) Whether or not these cases represent the vectorcardiographic counterpart of Burch s cases of septal fibrosis cannot be determined because post mortem proof is lacking in *the former However the* electrocardiograms in our cases showed neither Q waves in leads I aVL V and V_6 nor electrocardio*graphic or vectorcardiographic findings suggestive* of incomplete left bundle branch block or anteroseptal infarction The patients themselves were considered clinically to have coronary artery disease

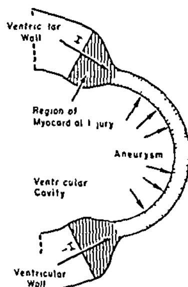
SUBENDOCARDIAL MYOCARDIAL INFARCTION

For reasons previously discussed injury and ischemia limited solely to the anterior subendocardium produce an S–T vector pointing to the right posteriorly and superiorly and mean instantaneous T spatial vectors directed anteriorly to the left and inferiorly and therefore project depressed S–T segments and upright T waves on left precordial leads In subendocardial infarction myocardial ischemia may be subendocardial as above or transmural in which case the mean instantaneous T spatial vectors are rotated posteriorly and cause anterior chest leads to register inverted T waves

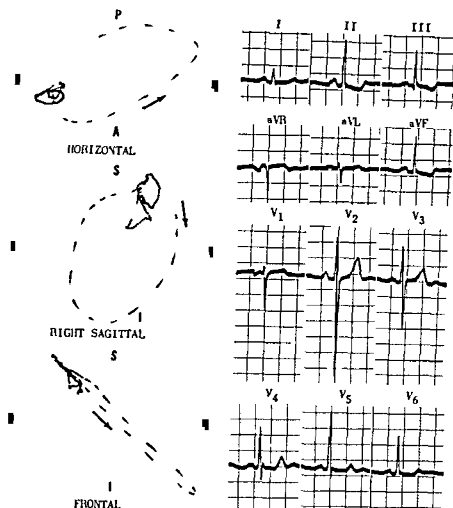
Since the S–T segment and T wave changes of subendocardial infarction are usually indistinguishable *from those of chronic coronary insufficiency the presence or absence of alterations in QRS configuration* assumes critical diagnostic significance Unfortunately the association of QRS changes (particularly abnormal Q waves) with subendocardial myocardial infarction has been the subject of some controversy The many studies of this problem have been summarized thus experimentally and clinically pathologic Q waves have been recorded in anterior precordial leads in some instances of subendocardial infarction but not in others Prinzmetal and his coworkers who believe the inner one third to two thirds of the ventricular wall to be electrocardiographically "silent" did not observe abnormal Q waves in acute or chronic subendocardial infarctions produced experimentally in dogs They concluded that "pure" subendocardial infarctions do not alter the QRS complex in the precordial electrocardiogram an opinion which is gaining considerable support The authors *of this text are in agreement with Prinzmetal's conclusions* At present the electrocardiographic status of subendocardial infarction remains unsettled and as far as is known there have been no vectorcardiographic descriptions of such an entity

VENTRICULAR ANEURYSM

Following an acute myocardial infarction the elevated S–T segments of the electrocardiogram ordinarily return to the isoelectric base line within several days to several months after onset of the acute episode However in a small number of cases the S–T segments remain upwardly displaced for many years or indefinitely and are usually associated with prominent Q waves or QS deflections Most of these patients are found to have had relatively massive infarctions previously In many but not all of these patients ventricular aneurysms can be demonstrated conversely some patients with ventricular aneurysms do not display persistent S–T segment elevations The mechanism responsible for the persistent injury



above The –I vectors project positive voltages on overlying leads during early electrical diastole and so the electrocardiogram registers elevated S–T segments (See text for detailed discussion of this mechanism)



I ,

normal septal Q waves are absent in leads I aVL V_5 and V_6 This finding in conjunction with the lack of evidence of anteroseptal myocardial infarction is strongly suggestive of fibrosis of the interventricular septum

The corresponding abnormality in the vectorcardiogram consists of the inscription of the initial portion of the QRS s£ loop to the left anteriorly and slightly superiorly There is nothing in the vectorcardiogram to suggest the presence of anteroseptal myocardial infarction or incomplete left bundle branch block *The abnormal direction of the initial segment of the QRS s£ loop is best explained by the presence of septal fibrosis In addition the T s£ loop is discordant to the long axis of the QRS s£ loop* The vectorcardiogram *therefore corroborates the electrocardiographic impression of septal fibrosis and also displays a nonspecific T s£ loop abnormality*

4 Q waves in leads V_5 and V_6 when there is complete left bundle branch block or in leads V_1 and V_2 when there is complete right bundle branch block Q waves in incomplete bundle branch block do not necessarily signify septal infarction

5 The sudden onset of left or right bundle branch block or of atrioventricular block during an episode clinically compatible with myocardial infarction

FIBROSIS OF THE INTERVENTRICULAR SEPTUM

Healed septal infarction can be simulated pathologically by coalescent foci of fibrosis in the septal muscle The diagnostic electrocardiographic abnormality which Burch has observed in cases of septal fibrosis consists of absence of normal septal Q waves *in leads I V and V_6 Normally the small Q waves* in these leads reflect initial septal activation in a left to right direction more precisely than does the initial R wave in lead V_1 which also results from depolarization of paraseptal portions of the left and right ventricular free walls *Consequently the presence* or absence of an initial R wave in lead V_1 would not be expected to correlate as well with the presence or absence of septal fibrosis as the presence or absence of Q waves in leads I V and V_6 In occasional vectorcardiograms we have observed that the horizontal and sagittal QRS loops were written initially to the left anteriorly and inferiorly but were otherwise normal in configuration and orientation (Fig 214) Whether or not these cases represent the vectorcardiographic counterpart of *Burch s cases* of septal fibrosis cannot be determined because post *mortem proof is lacking in the former* However the electrocardiograms in our cases showed neither Q waves in leads I aVL V and V_6 nor electrocardiographic or vectorcardiographic findings suggestive of incomplete left bundle branch block or anteroseptal infarction The patients themselves were considered clinically to have coronary artery disease

PERI INFARCTION BLOCK

In 1950 for the first time First Bayley and Bedford described a type of intraventricular conduction defect associated with myocardial infarction which they called *peri infarction block*. The identifying features of peri infarction block in the electrocardiogram are as follows (Fig 217)

1 The QRS interval is prolonged to 0 11 or 0 12 second
2 Onset of the intrinsicoid deflection in precordial leads overlying uninvolved portions of left ventricular wall occurs at the normal time but is delayed in leads overlying the peripheral zone of the infarction.
3 The electrocardiogram presents evidence of myocardial infarction If the latter is an anterior infarction, the electrocardiogram often has a superficial resemblance to left bundle branch block
4 As Grant has recently emphasized the initial and terminal QRS forces in peri infarction block are directed differently The spatial angle subtended by the initial and terminal 0 04 second mean QRS spatial vectors usually exceeds 60

The mechanism of peri infarction block is not known for certain although the mechanism outlined by First, Bayley and Bedford is generally albeit tentatively accepted by most authorities These authors postulate the following When myocardial infarction is accompanied or followed by onset of peri infarction block, it is presumed that the infarction has involved an extensive portion of the subendocardial muscle and Purkinje network As a result uninvolved muscle overlying the infarction cannot undergo activation in a direction perpendicular to the epicardial surface as occurs normally instead the activation wave must spread by a circuitous route through unaffected muscle at the periphery of the infarction The late activation of subepicardial muscle overlying the infarction is responsible in peri infarction block for the QRS prolongation for the delayed onset of the intrinsicoid deflection in leads oriented to the infarction and for the divergence of the initial and terminal instantaneous QRS vectors Thus for reasons previously explained the initial instantaneous QRS vectors are displaced away from the site of infarction and therefore away from the overlying exploring electrode of one or the other precordial lead On the other hand since the subepicardial muscle over the infarction is the last to be activated the terminal instantaneous QRS vectors are directed toward the exploring electrode of the same precordial lead Consequently in peri infarction block the lead in question records a QR or Qr deflection of prolonged duration

More recently Grant and his associates have described what they believe to be a type of peri infarction block different from that described above The principal point of distinction between the two types of peri infarction block according to Grant and his co-workers is the absence of significant QRS prolongation in the second type These investigators reviewed electrocardiograms recorded before and after onset of infarction in a large series of cases and found

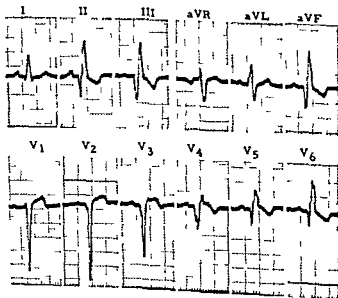


Fig 217 —Diaphragmatic and anterolateral myocardial infarctions in a man 55 with coronary artery disease The presence of peri infarction block is suggested by the tall and wide R waves in leads II III aVF V and V and the QRS duration of 0 12 second Peri

erence figure (the initial 0 04-second vector pointing *away from* the diaphragm and the terminal 0 04-second vector pointing *toward* the diaphragm) (*From* B S Lipman and E Massie *Clinical Scalar Electrocardiography* [4th ed. Chicago Year Book Publishers Inc 1959] Fig 191)

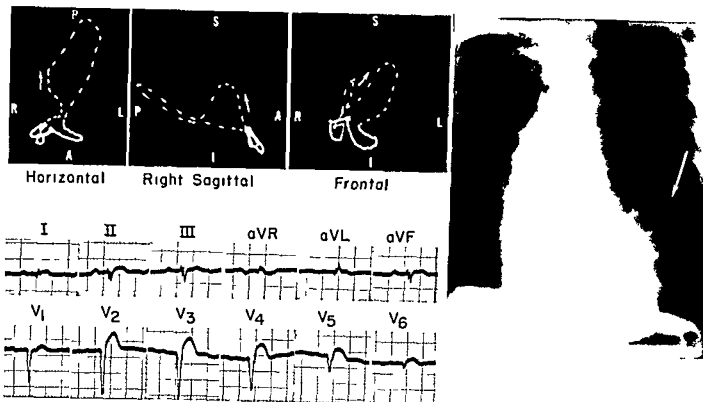


Fig 216 —Electrocardiographic and vectorcardiographic findings in a man 68 who later at postmortem was found to have had a ventricular aneurysm He had a confirmed history of *myocardial infarction* which occurred 1½ years before these recordings

The electrocardiographic findings of significance are a small Q wave followed by a small R wave in leads I and aVL

this text have no ready explanation for this feature although possibly it may reflect migration of [illegible] point The posteroanterior chest film of the patient is also reproduced above the arrow indicating the location of the large ventricular aneurysm The patient expired postoperatively after attempted surgical correction of the aneurysm

vector accompanying ventricular aneurysms is not definitely established but several possibilities have been entertained

1 *As the scarred thin walled aneurysm balloons* outwardly with each ventricular systole traction is exerted on the muscle to which it is attached This gives rise to injury currents in the surrounding myo cardium (Fig 215)

2 In some cases the S–T segment elevation may be secondary to deep *QS deflections (see discussion* of ventricular gradient in Chapter 6) since for every increase in the QRS area there must be an equal but oppositely directed increase in the area of the S–T interval and T wave

3 Grishman explains the persistence of upwardly displaced S–T segments following myocardial infarction by postulating a shift in position of the dipole center during inscription of the QRS loop due to a markedly disturbed balance of activation potentials Since after writing the QRS loop the electron beam of the vectorcardiograph does not return to its initial point of origin the QRS loop remains open the resulting S–T vector projecting S–T elevation on overlying leads The shift in position of the dipole center is said also to occur in the presence of *left bundle* branch block Here also the dipole shift is attributed *to the marked imbalance of electrical forces produced* by ventricular depolarization just as is the case with *myocardial infarction (Fig 216)*

PERI INFARCTION BLOCK

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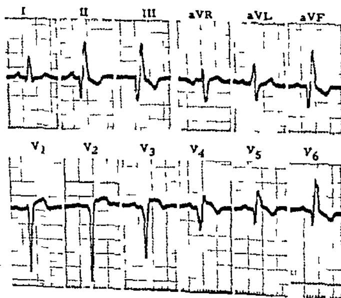
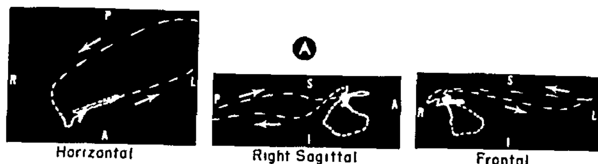
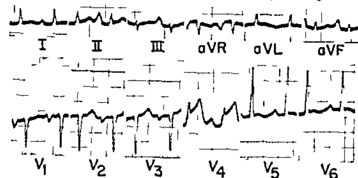
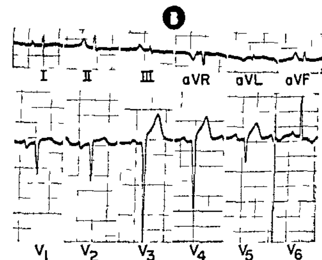


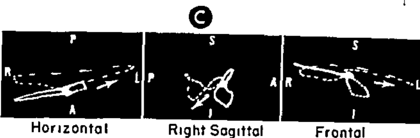
Fig 217 –Diaphragmatic and anterolateral myocardial infarctions in a man, 55 with coronary artery disease. The presence of peri infarction block is suggested by the tall and wide R waves in leads II III aVF V and V and the QRS duration of 0 12 second. Peri

...aphragm) (From B S Lipman and E Massie *Clinical Scalar Electrocardiography* [4th ed Chicago Year Book Publishers Inc 1959] Fig 191)









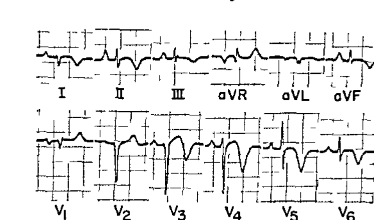


Fig 218 —*Peri infarction block complicating acute* anteroseptal myocardial infarction

In **A** the electrocardiographic findings of note are low initial R wave in lead V_1 QS deflections in leads V and V_3 markedly elevated S–T segments in lead V *and sl* version in myocardial cardiogran part of the efferent limb of the horizontal QRS loop and *because of the posterior deviation of the efferent* limb of the QRS sÊ loop the direction of inscription of the early portion of the right sagittal loop is reversed The QRS sÊ loop remains open indicating the presence of ai orly

ented slightly anteriorly and at first to the right and then to the left with the interpretation of the electrocardiographic bnormal t es

In the electrocardiogram in **B** recorded about deflections in leads V through V and low R wav vated but the T waves are upright Note that neit

The electrocardiogram in **C** was recorded severa day a e Lead wave and the T waves are inverted in leads I II III and aVF Leads aVR and V show terminal R waves while leads V_5 and V *both record deep terminal S waves The diagnostic findings of recent anterior myocardial infarction persist* the T waves being deeply inverted in leads V_2 through V_6 The vectorcardiogram recorded at this time differs from that in **A** primarily in two respects the QRS sÊ loop displays which is responsible for the terminal S waves appearing ir loop is large and directed to the right slightly anteriorly

that, in a s gnificant percentage of the cases antero-lateral infarctions and diaphragmatic infarctions in particular produced alterations in the terminal as well as the initial QRS vectors In most such cases ... vectors assumed relatively

... infarctions the altered terminal instanta... vectors generally were rotated to the left superiorly and posteriorly As Grant pointed out anterolateral infarction may therefore be implicated in occasional cases as the mechanism responsible for left axis deviation of A QRS Furthermore if QRS interval pro- ... superimposed on this type of terminal

tion) (Fig 218) Grant found the altered terminal QRS vectors usually oriented to the right posteriorly and inferiorly But sometimes the terminal vectors were directed to the right superiorly and anteriorly and projected terminal R deflections on leads V_{3R} and V_1 In such instances if QRS prolongation to 0 12 second supervenes the electrocardiogram may resemble right bundle branch block Grant suggested that possibly occasional infarctions may alter only the termi

haps to point out that strictly posterior infarction was not included in his study series This type of infarction produces abnormalities which are manifested relatively late in the QRS interval and the mechanism responsible for these findings may possibly be a form of peri infarction block The authors of this text and other investigators have noted vectorcardiographically that strictly posterior infarction quite characteristically alters either the initial and terminal or only the terminal instantaneous QRS vectors

Grant and his associates believe that anterolateral infarction with peri infarction block can usually be differentiated from left bundle branch block if the following facts are kept in mind

1 A Q wave in lead I with a duration greater than 0 02 second occurs only in peri infarction block never in uncomplicated left bundle branch block

2. A Q wave of less than 0 02 second duration may occasionally be present in lead V_6 in uncomplicated left bundle branch block but Q waves of 0 03 second duration or longer in this lead occur only in peri infarction block or in left bundle branch block with septal infarction

3 In the large series of cases of uncomplicated left bundle branch block studied by Grant and his co-workers an initial R wave was present in leads V_1 to V_4 in 45% of the cases the R wave was absent in lead V_1 alone in 35% and absent in leads V_1 and V in 15% However in only 5% of the cases of left bundle branch block was the initial R wave lost in leads V_1 through V_3 In none of the cases of left bundle branch block were QS deflections recorded as far to the left as lead V_4 In contrast initial R ... through

4 In ... R w ...

5 I ...

R waves decrease systematically in magnitude between leads V_1 and V_4 although this was not an unusual finding in peri infarction block

6 The best point of distinction between left bundle branch block and peri infarction block, according to Grant and his associates is the angle between the initial and terminal mean 0 04 second QRS spatial vectors In left bundle branch block the angle varies from 45 or less to 60 but in peri infarction block the angle is rarely less than 60 and usually exceeds 100

The primary difference between diaphragmatic myocardial infarction with peri infarction block and the condition it may resemble namely right bundle branch block is that the terminal 0 04 second mean QRS spatial vector although directed to the right in both types of conduction disturbance is situated as much as 90 more anterior in right bundle branch block than in peri infarction block In diaphragmatic myocardial infarction with peri infarction block the terminal mean vector lies slightly posterior and usually is directed more vertically than the corresponding vector in right bundle branch block

Grant and Murray are of the opinion that the type of peri infarction block observed in their infarction

series results from some mechanism other than that postulated by First, Bayley and Bedford. The following is a quotation from Grant and Murray:

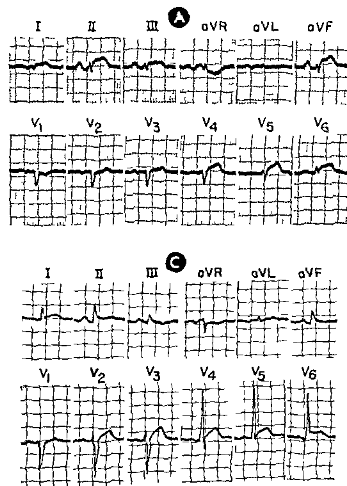
As a possible explanation for this type of peri-infarction block one may consider the conduction network in the left ventricle as consisting of two pathways which are effectively syncytial with one another over large areas of the myocardium. Under normal circumstances excitation spreads simult in
0.04 sec of the
spond perhaps

of the left bundle branch for which there is considerable anatomic and some physiologic evidence. Anterolateral infarcts tend to lie in the distribution of the *anterior subdivisions* and diaphragmatic infarcts tend to lie in the distribution of the posterior subdivisions. An infarct in one region would, if it involved a large enough portion of the network, cause excitation to spread largely by way of the other pathway so that excitation of the myocardium adjacent to the infarct would be delayed by 0.04 sec but there would be little or no prolongation of the QRS interval.

PERICARDITIS AND MYOCARDITIS

Pericarditis

Acute fibrinous pericarditis.—This condition injures the subepicardial myocardium and therefore produces an S–T vector directed toward the effective location of the epicardial injury. For example, pericarditis involving the anterior aspect of the left ventricle projects elevated S–T segments on anterior precordial leads. The T waves are usually upright during this phase. With subsidence of the acute pericarditis the elevated S–T segments begin to return to the base line and inverted T waves make their appearance. In all probability the latter are due to an abnormally delayed onset of repolarization in the subepicardial myocardium secondary to the myocardial inflammatory response. Accordingly the T sE loop and sA T tend to swing away from the involved area. If there are no complications all abnormalities disappear from the electrocardiogram within 4–8 weeks. When pericarditis occurs in adults within age groups prone to coronary artery disease, S–T segment deviation due to pericarditis and that occurring in early stages of developing myocardial infarction may be difficult to differentiate electrocardiographically, crucial as such a



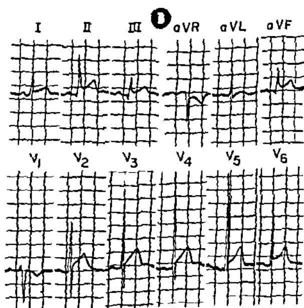


Fig. 219.—A–C, three different electrocardiographic examples of a relatively early stage in the evolution of the pericarditis pattern. Note in each record the widespread S–T segment elevation occurring in leads I, II, III, aVF and V (or V_1) through V, and the normal T waves. Later in the evolution of changes the S–T segments will return to the isoelectric lines, and as they do so the T waves will become progressively lower and finally inverted.

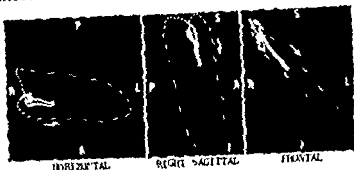
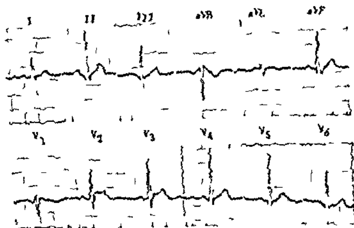


Fig 220 – Electrocardiographic and vectorcardiographic findings in subacute



from striking in these leads

In the vectorcardiogram the QRS sE loop remains open, its terminus being displaced slightly anteriorly and to the left. The S–T vector extending from the electrical null point to the terminus of the QRS E loop in each projection is compatible with anterolateral subepicardial injury such as occurs in pericarditis

distinction is to the prognosis and therapeutic management of the patient. Serial electrocardiograms are indicated, therefore, to distinguish between the two clinical possibilities. In interpreting the initial electrocardiogram recorded from such a patient, two features tend to s ... th ...

1 ... t

cardius than in myocardial infarction in that the subepicardial injury pattern does not conform to a single area of involvement (in terms of the specific leads displaying elevated S–T segments). For example, one often observes S–T segment elevation in leads I, II, III, aVL and aVF and in most of the precordial leads. Such a distribution of S–T segment elevation would be quite unusual in myocardial infarction (Figs 219 and 220).

2 The association of upright T waves and elevated S–T segments is more frequently encountered in pericarditis than in myocardial infarction. In the subepicardial injury phase of infarction the T waves are usually difficult to separate from the S–T segments or they are of low amplitude or are terminally inverted.

Pericarditis with effusion – This is characterized by lowered voltage of all components of the ventricular complex. The low amplitude QRS and T complexes may reflect a short-circuiting of the cardiac potentials by the pericardial effusion.

Chronic constrictive pericarditis – The electrocardiogram in this condition may display the following:

1 Low voltage of all components of the ventricular complex due possibly to a decreased volume of ventricular muscle secondary to prolonged reduction in work requirements
2 Unusually wide angle between sÂ QRS and sÂ T

Myocarditis

Various forms of myocarditis, including *acute rheumatic carditis*, may produce one or more of the following abnormalities:

1 Conduction disturbances such as prolonged Q–T intervals, atrioventricular block and bundle branch block
2 Disturbances in the cardiac rhythm such as sinus tachycardia, atrioventricular nodal rhythms with atrioventricular dissociation and occasionally atrial fibrillation
3 An abnormally wide spatial angle between sÂ QRS and sÂ T
4 The electrocardiographic abnormalities described in the discussion of acute fibrinous pericarditis or pericarditis with effusion

Myocardial Damage, Coronary Insufficiency, and Stress Tests

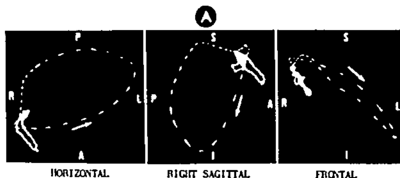
MYOCARDIAL DAMAGE AND CORONARY INSUFFICIENCY

WITH THE EXCEPTION of the rare infarctions resulting from congenitally aberrant or anomalous coronary arteries or from syphilitic involvement of the coronary ostia a myocardial infarction generally indicates underlying coronary arteriosclerosis or atherosclerosis However many patients with coronary artery disease never sustain clinically recognizable infarctions although after recurrent episodes of myocardial anoxia they may eventually suffer diffuse degenerative and fibrotic changes in the ventricular myocardium In these patients as well as in those who have had small subclinical myocardial infarctions the vectorcardiogram may display only minor deviations of the QRS sÊ loop from normal For example the contour of the QRS sÊ loop may be markedly irregular with abrupt changes in course which reflect sudden changes in the direction and magnitude of the mean instantaneous QRS spatial vectors Sometimes the direction of inscription of the QRS sÊ loop may be reversed in one or more projections of the vectorcardiogram not infrequently the QRS sÊ loop may have a markedly irregular contour and or may not lie in a single plane of predilection or finally S–T vector or T sÊ loop abnormalities may be present in the vectorcardiogram With reference to the latter the authors of this text consider an angular divergence of the long axes of the QRS sÊ and T sÊ loops greater than 45 to be the vectorcardiographic equivalent of nonspecific T wave abnormalities in the electrocardiogram However these vectorcardiographic abnormalities may or may not be accompanied by recognizable abnormalities of the QRS deflections S–T segments or T waves of the electrocardiogram (Figs 221–223) When corresponding QRS changes are present in the electrocardiogram they may take for example the form of notched or slurred QRS complexes low R waves of equivocal significance in one or more precordial leads or Q waves which are suspicious but not definitely abnormal In general the QRS sÊ loop abnormalities reflecting myocardial damage and corresponding abnormalities in the electrocardiogram have not been studied extensively as yet

Much more attention has been given to the S–T segment and T wave or T sÊ loop changes in coronary artery disease In some patients these abnormalities may remain as a persistent feature of the electrocardiogram or vectorcardiogram while in others they may appear only during episodes of angina pectoris or after exercise or exertion The S–T segment and T wave abnormalities may be grouped as follows

Subendocardial ischemia and injury—The electrocardiographic and vectorcardiographic patterns of subendocardial ischemia and injury are considered to be most typical of coronary insufficiency Subendocardial myocardial injury produces an S–T vector directed to the right posteriorly and superiorly away from the effective electrical site of subendocardial injury in the left ventricle while ischemia limited to subendocardial layers of muscle rotates the mean instantaneous T vectors in just the opposite direction—that is toward the electrical location of the ischemic muscle Thus subendocardial injury and ischemia are evidenced in the electrocardiogram by the presence of depressed S–T segments and upright T waves in left precordial leads

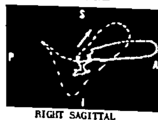
Transmural ischemia—When ischemia extends through the entire thickness of ventricular wall to the epicardial surface the mean instantaneous T vectors

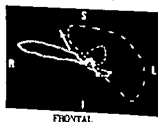


I II III aVR VL VF

V_1 V_2 V_3 V_4 V_5 V_6







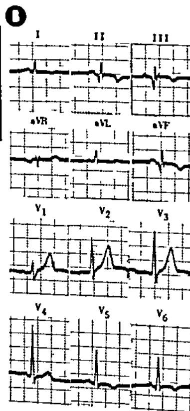


Fig 221 —A electrocardiogram and vectorcardiogram recorded from a patient with a history of angina pectoris. Although the electrocardiogram is within normal limits the QRS $s\hat{E}$ and T $s\hat{E}$ loops of the vectorcardiogram are abnormally divergent. Thus the spatial angle subtended by the long axes of the QRS $s\hat{E}$ and T $s\hat{E}$ loops (calculated by the method described in Chapter 7) is approximately 72 while the upper limits of normal is 45 This vectorcardiographic abnormality is compatible with myocardial disease or coronary artery disease The foregoing interpretation of this vectorcardiogram is substantiated by the subsequent course of events for 2 weeks later the patient was admitted to the hospital with the clinical picture of acute myocardial infarction

B electrocardiogram and vectorcardiogram recorded shortly after the patient's admission Both are diagnostic of acute diaphragmatic and probable posterolateral myocardial infarction.

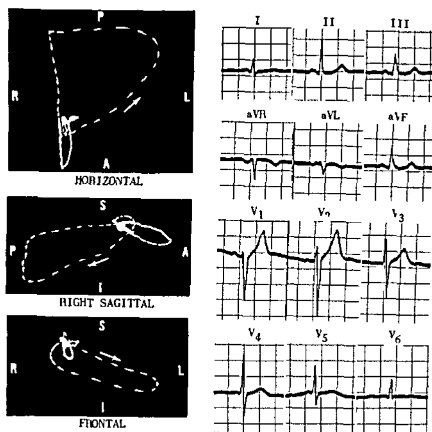


Fig 222 —Electrocardiographic and vectorcardiographic findings in a patient with typical angina pectoris The main abnormality in the electrocardiogram is the low T wave in lead I and the low to diphasic T wave in lead V_6 However these findings are far from impressive In contrast in the vectorcardiogram the spatial angle subtended by the long axes of the QRS sÊ and T sÊ loops is approximately 110 which is more than twice normal

swing away from the electrical site of ischemia Ordinarily this causes increased divergence of the mean instantaneous QRS vectors and mean instantaneous T vectors with the result that leads registering upright QRS deflections tend to record T waves which become gradually lower isoelectric and then inverted with progression of the ischemia In coronary artery disease transmural ischemia may occur in company with subendocardial injury or may be present in the absence of current of injury This pattern may be persistent or evanescent

Local transmural injury —Occasionally a local subepicardial injury pattern in the form of S–T segment elevations may appear over one aspect of the heart A temporary pattern of diaphragmatic wall injury is frequently observed

STRESS TESTS

Unfortunately for the physician the diagnosis of coronary artery disease is often difficult to establish particularly in those patients who have atypical anginal pain and a normal resting electrocardiogram In such situations it is frequently helpful diagnostically to utilize either the exercise test or the anoxemia test The latter procedure is probably less widely used than the exercise test because it requires special equipment However neither test possesses any definite advantage over the other although it is of some interest that patients having negative exercise tests may have positive anoxemia tests and vice versa These tests are undertaken only if control electrocardiographic tracings recorded just before commencing the tests show no significant abnormality and provided the patient is not receiving digitalis or other medications known to alter the duration of the excited state in heart muscle

The Anoxemia Test

For the anoxemia test Levy and his co workers recommend the use of a mixture of 10% oxygen and 90% nitrogen which is inspired by the patient at a rate comparable to that of normal pulmonary ventilation A control electrocardiographic tracing is obtained before beginning the test Thereafter records are taken at 5 minute intervals during the test which unless chest pain or discomfort interrupts usually lasts 25 minutes The criteria of Levy and his associ

ates for an abnormal test response are as follows
1 Arithmetical sum of the S–T deviations in all four leads (I II III and IV_F*) totaling 3 mm or more
2. Partial or complete reversal of the direction of the T wave in lead I accompanied by an S–T deviation of 1 mm or more in this lead
3 Complete reversal of the direction of the T wave in lead IV_F with or without S–T segment deviations

(For further information the reader is referred to the thorough review of this test by Stewart and Carr)

The Exercise Test

The purpose of this procedure is to increase by means of exercise the demands placed on the coronary blood supply of the myocardium If the ability of the coronary blood flow to adjust to the increased needs of the heart is limited because of pathologic narrowing of the coronary arteries relative ischemia and/or injury may be induced by exceeding this limit and electrocardiographic abnormalities will then appear

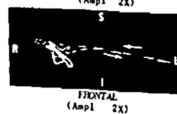
The recognition of an abnormal electrocardiographic response to exercise is made considerably more difficult by certain changes which normally occur with exercise These changes principally involve the S–T segment and the T wave With reference to the former the normal S–T segment deviations which are observed in healthy subjects may be subdivided into two groups which in this text will be called *apparent S–T deviations* and *physiologic S–T deviations* These deviations will be discussed in the paragraphs to follow

* Lead IV_F is an obsolete bipolar precordial lead The recording positive electrode is applied at the same point as the exploring electrode in lead V and the negative electrode is attached to the left leg









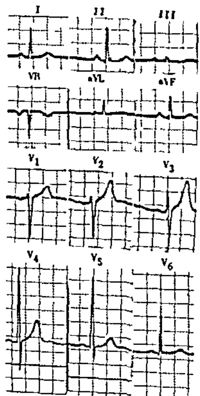


Fig 223 —Electrocardiographic and vectorcardiographic findings in a patient with typical angina pectoris The electrocardiogram is within normal limits although there is a tendency to high QRS voltage in lead V but in the vectorcardiogram the spatial angle between the QRS sÊ and T sÊ loops is 85 well above the upper limit of normal

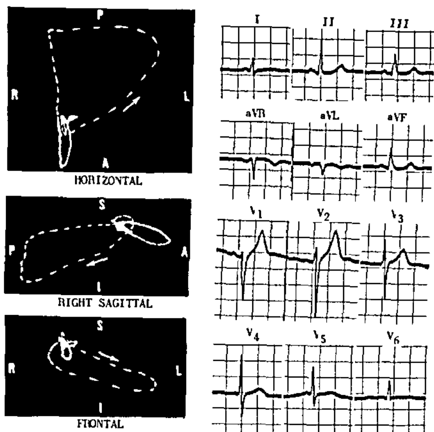


Fig 222 —*Electrocardiographic and vectorcardiographic findings in a patient with typical angina pectoris The main abnormality in the electrocardiogram is the low T wave in lead I and the low to diphasic T wave in lead V However these findings are far from impressive In contrast in the vectorcardiogram the spatial angle subtended by the long axes of the QRS sE and T sE loops is approximately 110 which is more than twice normal*

swing away from the electrical site of ischemia Ordinarily this causes increased divergence of the mean instantaneous QRS vectors and mean instantaneous T vectors with the result that leads registering upright QRS deflections tend to record T waves which become gradually lower isoelectric and then inverted with progression of the ischemia In coronary artery disease transmural ischemia may occur in company with subendocardial injury or may be present in the absence of current of injury This pattern may be persistent or evanescent

Local transmural injury —Occasionally a local subepicardial injury pattern in the form of S–T segment elevations may appear over one aspect of the heart A temporary pattern of diaphragmatic wall injury is frequently observed

STRESS TESTS

Unfortunately for the physician the diagnosis of coronary artery disease is often difficult to establish particularly in those patients who have atypical anginal pain and a normal resting electrocardiogram In such situations it is frequently helpful diagnostically to utilize either the exercise test or the anoxemia test The latter procedure is probably less widely used than the exercise test because it requires special equipment However neither test possesses any definite advantage over the other although it is of some interest that patients having negative exercise tests may have positive anoxemia tests and vice versa These tests are undertaken only if control electrocardiographic tracings recorded just before commencing the tests show no significant abnormality and provided the patient is not receiving digitalis or other medications known to alter the duration of the excited state in heart muscle

The Anoxemia Test

For the anoxemia test Levy and his co workers recommend the use of a mixture of 10% oxygen and 90% nitrogen which is inspired by the patient at a rate comparable to that of normal pulmonary ventilation A control electrocardiographic tracing is obtained before beginning the test Thereafter records are taken at 5 minute intervals during the test which unless chest pain or discomfort interrupts usually lasts 25 minutes The criteria of Levy and his associ

heart during exercise This factor has been implicated, by some investigators in the production of the tall upright T waves often seen after exercise. The presence or absence of sympathetic stimulation during an episode of tachycardia is probably important in deciding whether ST-T wave changes appear although its influence in a given case is difficult to assess

Abnormal ECG Responses to Exercise

The crux of the problem relating to the interpretation of the effects of exercise on the electrocardiogram les in differentiating S-T segment and T wave [illegible]

[illegible] pine criteria of abnormality If the criteria employed are quite strict, then a positive response to the test constitutes strong evidence for coronary artery disease, but at the same time a large number of false negative test responses must be anticipated *On the* other hand, if the test is to be used to eliminate the possibility of coronary artery disease false negative responses must be excluded, in so far as possible by utilization of less rigid criteria of abnormality However less rigid test criteria have the associated disadvantage of eliciting many false positive test responses in normal subjects Thus the criteria selected should be those best suited to the purpose for which the stress test is being done Scherf and Schaffer prefer for their purposes a rigid approach to the exercise test.

Criteria for abnormality —The electrocardiographic criteria for an abnormal exercise test proposed by different authorities are as follows

1 An S-T segment depression greater than 2 mm in any lead is conceded, by all investigators to be abnormal
2 An S-T segment depression of 1 5–2 mm is considered probably abnormal but if it is less than 1 5 mm Scherf and Schaffer regard it as normal realizing that in some instances false negative test responses result.
3 Twiss and Sokolow designate as the upper limit of normal, an S-T segment depression of 1 mm in lead I and of 1.5 mm in leads II and III Biorck regards as *normal* those S-T segment depressions whose total sum in the three standard limb leads is less than 2 mm while Master and his associates accept a 0.5-mm deviation of the S-T segments in any lead as the upper limit of normal
4 The appearance of a pattern of local subepicardial injury as previously described is abnormal
5 Abnormal widening of the angle between sÂ QRS and sÂ T produces positive findings of T wave inversion in leads registering upright QRS complexes such as leads I II and V_4 through V_6 If the T wave becomes lower or inverted at a time when the postexercise *tachycardia* is subsiding this observation carries increased significance in terms of abnormality
6 The occurrence of extrasystoles after exercise is thought to be definitely abnormal They are usually ventricular in type and may be multifocal in origin Occasionally paroxysmal tachycardia may be precipitated by exercise
7 Onset of significant atrioventricular or intraventricular conduction delay after exercise is a fairly reliable sign of coronary artery disease

Technique of the Exercise Test

Inasmuch as strenuous exercise in normal subjects often elicits marked electrocardiographic changes the criteria cited above are applicable only if the subject performs moderate exercise *Moderate exercise* is considered [illegible]
moderate p [illegible]
equivalent [illegible]
given perso [illegible]
in one of three ways as follows

1 On the basis of the history obtained from the patient Scherf and Schaffer prescribe an amount of exercise not exceeding that which the patient permits himself to perform daily and approximating that which usually causes the patient to experience angina or its equivalent.

2 Other authorities have the patient exercise until he is stopped by pain dyspnea or fatigue Scherf and Schaffer stress that this test inherently carries an increased danger to the patient and therefore should be reserved for cautious use in those patients with negative responses to more conservative testing

3 Perhaps the most popular method at present is based *on the two-step test of Master*

The Master Test

The Master steps usually consist of a wooden platform having two steps 9 inches wide and 9 inches from the floor placed on each side of a central step 9 inches wide and 18 inches from the floor When the patient climbs up one side of the steps and down the other he has completed one trip Master has devised tables which relate the number of trips to be taken

Apparent S–T Segment Deviations

In the presence of marked tachycardia an apparent S–T depression may be due to an elevation of the T–P segment which is itself the result of superimposition of the P wave on the T wave of the preceding complex

Another cause of apparent S–T segment deviation after exercise is superimposition of a prominent auricular repolarization wave (Ta wave) on the ventricular S–T segment The Ta wave lasts well beyond the end of the QRS complex even though the P–Ta interval is shortened by tachycardia Therefore an increase in the size of the Ta deflection may displace *the ventricular S–T segment downward (or upward* if the Ta wave follows an inverted P wave and is of positive polarity) Such an increase in the magnitude of the Ta deflection in exercise is probably caused by two factors

1 The mean P wave vector becomes more vertical with exercise and so there is an increased size of the P waves in leads II and III particularly Since the area of the P wave *is equal in size but opposite in* polarity to the area of Ta an increase in the size of an upright P wave causes an increase likewise in the size of the negative Ta wave

2 More important is the fact that tachycardia shortens the monophasic action current of the atrial myocardium affecting chiefly the recovery phase during which the Ta wave is written As the Ta wave shortens it must also deepen in order to maintain an area equal and opposite to that of the P wave

Some correction for the foregoing two mechanisms producing apparent S–T segment deviations can be accomplished in the following ways (*a*) The level of the S–T segment can be compared to the level of the point of junction of the P–R segment with the QRS complex (*b*) Occasionally deviation of the S–T segment by a prominent Ta wave can be demonstrated more clearly by extending the downwardly sloping P–R segment into the S–T segment If this sloping line descends to the level of the S–T segment then apparent S–T segment deviation due to a large Ta wave is probably present (*c*) The recording of lead V_4 or V may often be helpful since in this lead the P–Ta complex is usually small

Physiologic S–T Segment Deviations

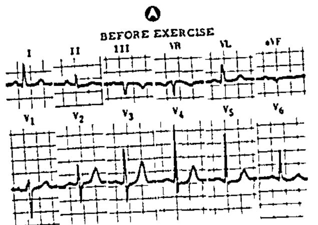
In a previous section it was stated that normally there is a difference in the duration of the excited state between inner and outer areas of the ventricular myocardium that is to say normally there exists a ventricular gradient The vector representing this gradient like the mean ST–T vector * tends to parallel the mean QRS vector Theoretically if the duration of the excited state or the degree of repolarization delay were the same in all parts of the ventricular myocardium the ventricular gradient would be zero and the mean QRS and ST–T vectors would be of equal amplitude and directed 180 away from each other In reality the ventricular gradient is never zero but as it decreases the ST–T vector rotates away from the mean QRS vector Thus electrocardiographic leads registering upright QRS complexes may *develop S–T segment* depression and *T wave flattening* or inversion which tend to increase as the ventricular gradient approaches zero There are two factors related to the ventricular gradient which act to produce a physiologic S–T segment deviation after exercise in normal individuals

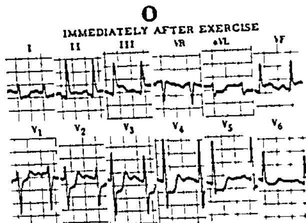
1 Normally an increase in the heart rate results in a decrease in the ventricular gradient Apparently at faster heart rates the inner layers of the myocardium normally the site of repolarization delay tend to recover more promptly Consequently as *their speed* of recovery approaches that of the outer layers the duration of the excited state becomes more nearly equal at the endocardial and epicardial surfaces of the heart the ventricular gradient approaches zero and the S–T segments and T waves descend

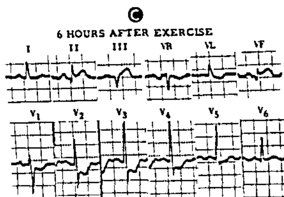
2 As with the P–Ta waves in shortening the monophasic action current of the ventricular myocardium tachycardia shortens mainly the recovery process and this produces a shorter Q–T interval In order to maintain its same area the ST–T complex must increase its amplitude or area becoming more positive or more negative depending on whether it follows a resultantly upright or inverted QRS complex

It should be added that an effort" factor is also thought to be present in exercise which to a varying degree opposes the tendency of the above tachycardia factor to decrease the ventricular gradient The effort factor possibly functions by delaying repolarization in the inner layers of the myocardium as the result of the more intense mechanical effort of the

The electrical forces normally produced during both the S–T interval and the T interval result from ventricular repolarization Consequently factors such as those to be described which influence the repolarization process tend to manifest similar effects on both the S–T segment and the T wave For this reason these two components *of the QRS–T complex* will be referred to occasionally as the *ST–T complex* or as being represented by an *ST–T vector*







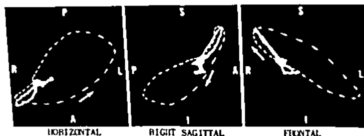


Fig 224 – Electrocardiographic and vectorcardiographic findings during and after a double Master exercise test

The control electrocardiogram in **A** is not entirely within normal limits in that there is definite although slight S–T segment depression in leads V through V. Since the patient was not on digitalis medication, the S–T segment deviation in the electrocardiogram would have to be considered suggestive of subendocardial myocardial injury. With rare exceptions an abnormal control electrocardiogram renders the exercise test purposeless and potentially hazardous.

The electrocardiogram in **B** recorded immediately after exercise displays marked S–T segment depression in leads I, aVL, and V through V and equally marked S–T segment elevation in leads II, III, and aVF. The latter finding is indicative of diaphragmatic subepicardial myocardial injury. The S–T segment depression in the anterior precordial leads can be attributed to either or both of the following factors: anterior subendocardial myocardial injury and/or posterior subepicardial myocardial injury (the precordial leads recording reciprocal S–T segment deviation).

In the electrocardiogram in **C** recorded 6 hours after the exercise stress test, abnormal Q waves have appeared in leads III and aVF and S–T segment elevation persists in these leads and in lead II. The S–T segments in leads V through V remain depressed. The abnormalities present in this electrocardiogram are diagnostic of acute diaphragmatic myocardial infarction. The vectorcardiogram in **C** was recorded several days after the recording of the electrocardiogram appearing above it. The QRS sÊ loop displays a large early rightward and superior deflection, the mean 0.02-second instantaneous vectors of the right sagittal and frontal QRS loops being situated superior to −40. These QRS sÊ loop abnormalities are diagnostic of diaphragmatic myocardial infarction while the anterior, rightward, and superior orientation of the T sÊ loop is indicative of diaphragmatic posterolateral myocardial ischemia.

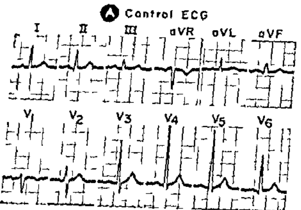
TABLE 28 —NUMBER OF TRIPS PERFORMED DURING 1½ MINUTES IN THE MASTER TWO STEP EXERCISE TEST*

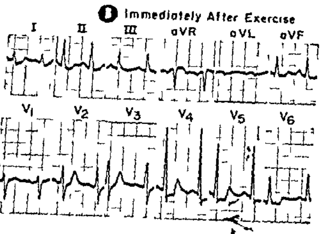
Males (Females in Parentheses)

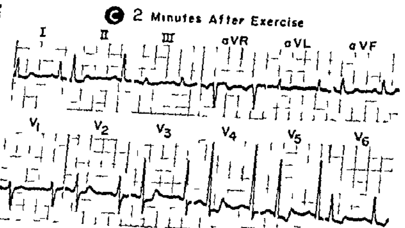
Weight	Age in years							
	5–9	10–14	15–19	20–29	30–39	40–49	50–59	60–69
lb	♂ = ♀							
40–49	35	36 (33)	(33)					
50–59	33	35 (33)	32 (32)					
60–69	31	33 (32)	31 (30)					
70–79	28	32 (30)	30 (29)					
80–89	26	30 (28)	29 (28)	29 (28)	28 (27)	27 (24)	25 (22)	24 (21)
90–99	24	29 ([illegible])	28 ([illegible])	28 ([illegible])	[illegible] (25)	26 (23)	25 ([illegible])	23 (20)
100–109	22	27 ([illegible])	27 (25)	28 (26)	27 (25)	25 (23)	24 (21)	22 (19)
110–119	20	26 (23)	[illegible] (23)	[illegible] (25)	26 (24)	25 (22)	23 (20)	22 (18)
120–129	18	24 (22)	25 ([illegible])	26 (24)	26 (23)	24 (21)	23 (19)	21 (18)
130–139	16	23 (20)	24 (20)	25 (23)	25 ([illegible])	23 (20)	22 (19)	20 ([illegible])
140–149		21 (18)	23 (19)	[illegible] ([illegible])	[illegible] (21)	23 (19)	21 (18)	20 (16)
150–159		20 (17)	22 ([illegible])	24 (21)	24 (20)	[illegible] (19)	20 ([illegible])	19 (16)
160–169		18 (15)	21 (18)	23 (20)	23 (19)	22 (18)	20 (16)	18 (15)
170–179		(13)	20 (14)	22 ([illegible])	23 (18)	21 (17)	19 (16)	18 (14)
180–189			19 (13)	21 (18)	22 ([illegible])	20 (16)	19 (15)	17 (14)
190–199			18 (12)	21 (17)	21 (16)	20 (15)	18 (14)	16 (13)
200–209				20 (16)	21 (15)	19 (14)	17 (13)	16 (12)
210–219				19 (15)	20 (14)	18 (13)	17 (13)	15 (11)
220–229				18 (14)	20 (13)	18 (13)	16 (12)	14 (11)

*Reproduced by permission from Master, M. A.: The electrocardiogram after exercise. Standardized heart function test. U. S. Navy M. Bull. 40: 346, 1942.

in a 1½ minute interval to the subject's sex, age and weight (Table 28). Electrocardiographic tracings are taken before, immediately after and at 2, 4, 6 and 10 minute intervals after the test. If the response from the single Master two step test is negative, a double test may be performed later by having the subject complete twice as many trips in a 3 minute interval. When the physician feels that the patient is able to tolerate this amount of exercise, a double two step Master test is often done initially. At the end of each trip, the subject should always turn in the same direction (e.g., toward the wall) to avoid dizziness. In addition, the subject should be instructed to notify the physician present at the first onset of chest pain or undue dyspnea or fatigue, because at this point the test should be terminated (Figs. 224–227).







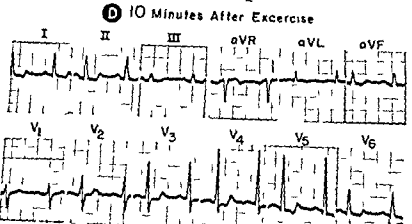
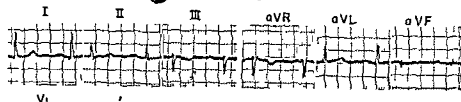
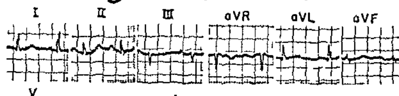
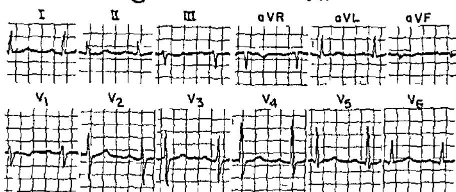


Fig. 226.—Positive response to double Master test. Record **A** shows normal control electrocardiogram. Record **B**, made immediately after exercise, displays S-T segment depression in leads I, II, aVF and V through V. In leads V and V, particularly, the S-T segment depression exceeds 1.5 mm. These abnormalities persist, although diminished in degree, for 10 minutes after exercise (records **C** and **D**).







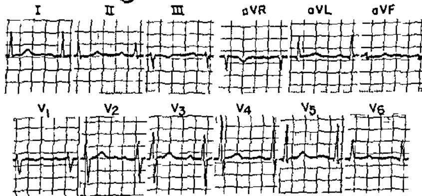
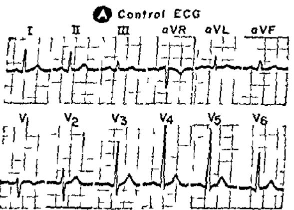
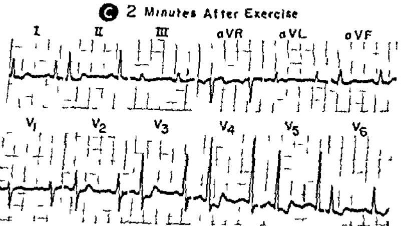
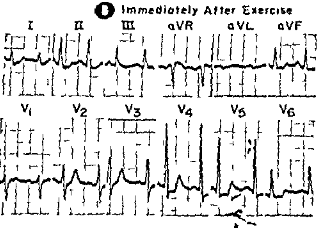


Fig 225 —Negative response to double Master test The apparent depression of the S–T segments in lead II of record B can most certainly be attributed to a superimposed deep atrial T wave (Ta wave) since the downward slope of the P–R segment can be continued into the junction of the QRS deflections and S–T segments







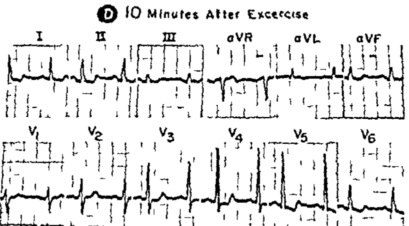


Fig. 226.—Positive response to double Master test. Record **A** shows normal control electrocardiogram. Record **B**, made immediately after exercise, displays S–T segment depression in leads I, II, aVL and V through V. In leads V and V particularly, the S–T segment depression exceeds 1.5 mm. These abnormalities persist, although diminished in degree, for 10 minutes after exercise (records **C** and **D**).

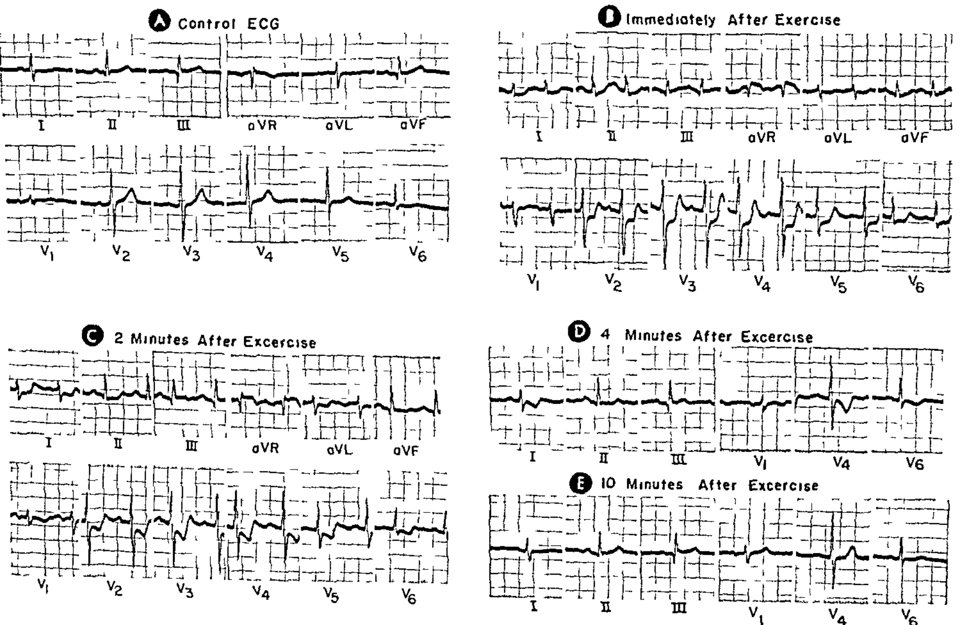


Fig 227 –Positive response to single Master test in a patient with a control electrocardiogram which is probably abnormal The electrocardiogram in **A** shows low to flat T waves in leads I and V suspiciously prominent Q waves in leads III and aVF and a low amplitude almost equiphasic RS deflection in lead V The tracing in **B** shows marked S–T segment depression in the precordial leads while subsequent record **C** displays inverted T or diphasic T waves in leads V through V which finally disappear 10 minutes after exercise (**E**) An electrocardiogram and vectorcardiogram recorded from this patient several months after the exercise stress test showed diagnostic findings indicating diaphragmatic posterolateral infarction in the past

PART III

The Cardiac Arrhythmias

Disturbances of Impulse Formation and Conduction General Considerations

GENERAL INTRODUCTION

THE PROPERTY OF automatic impulse formation is not confined to a single cardiac focus but is shared by nodal and Purkinje tissues scattered throughout the

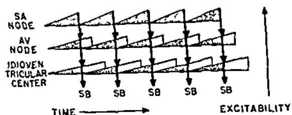


Fig
three
time
pacemakers discharges spo
th period of time required by the atrio-

even slower than that in the atrioventricular node It fol lows therefore that the sinus node will keep in a state of suppression all other pacemaking centers in the heart

heart Every focal collection of nodal or Purkinje cells has the potentiality of functioning as a cardiac pace maker but if all rhythmical centers were active si multaneously the result would be chaotic heart action Efficient cardiac function requires that all parts of the heart respond to a single rhythm and this in turn implies that one potential pacemaker must dominate all others For a rhythmical focus to emerge as domi nant pacemaker its impulses must be formed dis

charged and conducted so rapidly through the heart that all other centers of slower impulse formation are k ... charged state

ward (in descending order tail of the sinoatrial node coronary sinus area upper middle and lower portions of the atrioventricular node bundle of His and finally the more distal parts of the intraventricular conduction system and Purkinje network) (Fig 228) The graded rhythmicity of the impulse forming cen ters not only enables a single dominant pacemaker normally the sinoatrial node to drive the heart but also provides a mechanism by which suppression of one pacemaker calls into action the potential pace

be less susceptible to neurogenic depression than is the sinus node As a result widespread and uniform inhibition of all rhythm centers does not ordinarily occur except terminally

Cardiac rhythms (often referred to as *ectopic rhythms*) which originate in some focus other than the sinoatrial node are divided for purposes of dis cussion into the two following groups according to *their manner of origin*

Escape beats or rhythms —Depressed impulse for *mation in* or *blocked impulse* conduction from a higher pacemaker may permit a lower center to "es cape" *for one* or more beats at its own inherent rhythmicity and rate (Fig 229) Beats or rhythms so

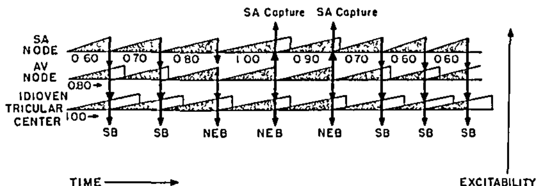


Fig 229 – The mechanism of escape beats This illustration differs from Figure 228 in that the time required by the sinoatrial node to reach its excitability threshold varies sometimes being short and at other times much longer As the rate of impulse formation slows in the sinoatrial node it eventually falls below the inherent rate of impulse formation in the atrioventricular nodal pacemaker at which time the atrioventricular node escapes to produce a nodal escape beat (*NEB*) In the present figure the atrioventricular nodal impulse not only prematurely discharges or captures the sinoatrial node (SA capture) and in so doing activates the atria but it also activates the ventricles and discharges prematurely the idioventricular center Consequently for two cycles of the cardiac rhythm the atrioventricular nodal center is the dominant pacemaker It should be noted that the atrioventricular node escapes at its own inherent rate of impulse formation because of slowing of impulse formation in a higher center An entirely different situation exists in Figure 230

produced are called in this text escape beats or *rhythms*

Ectopic beats or rhythms – The term *ectopic* as used hereafter is applied only to those beats or rhythms arising as the result of some change in excitability locally in the region of impulse origin (Fig 230)

The mechanism and electrocardiographic features of cardiac arrhythmias are related to the manner in which impulses are not only formed but also conducted The following paragraphs are devoted to a preliminary discussion of the refractory period of heart muscle and its relationship to interference and dissociation and to blocked impulse conduction

Once heart muscle has undergone excitation it remains for a period of time completely nonresponsive to impulse stimuli This interval the *absolute refractory phase* is followed by the *relative refractory phase* during which the muscle cells gradually recover their excitability and conductivity (Fig 231) Impulses entering the muscle tissue during the relative refractory phase may be conducted normally slowly incompletely or not at all depending on the following variables (*a*) the time of arrival of the impulses whether early or late in the relative refractory phase and (*b*) the relationship between impulse strength and the excitability threshold of the muscle

The absolute and relative refractory phases are of approximately the same duration and their total length equals the duration of the refractory period The length of the refractory period varies directly with the cycle length of the cardiac rhythm the longer the preceding cycle the more prolonged the *refractory period and the shorter the cycle the* shorter the refractory period However there is a lower limit beyond which further shortening of the cycle is not accompanied by parallel changes in the refractory period

The duration of the refractory period is normally longest in atrioventricular junctional tissues Therefore in a sense the atrioventricular node is the weak link in the chain of conducting pathways between the sinus node and the Purkinje system in the ventricles Because of their normally slow rate of conduction and subsequent recovery the atrioventricular junctional tissues are readily depressed by drugs or disease processes which lengthen the refractory period of heart muscle even though conduction elsewhere in the heart is not detectably disturbed A fact to be kept in mind is that atrioventricular (and sinoatrial) junctional tissues are capable of bidirectional conduction This property is best exemplified by beats *originating in the atrioventricular node* The nodal impulse not only passes in a forward or antegrade direction to activate the ventricles but also travels up the atrioventricular node and spreads over the atria in a retrograde direction That retrograde sinoatrial conduction may also occur is demonstrated whenever an ectopic atrial or nodal impulse prematurely discharges the sinus pacemaker Inasmuch as variations in antegrade and retrograde atrioventricular conduction are encountered relatively frequently in clinical electrocardiography as manifestations of atrioventricular interference or atrioventricular block these

mechanisms will be briefly discussed in this chapter and developed later in more detail

Atrioventricular Interference and Dissociation

The term *interference* is applied to the mechanism involved when conduction of one impulse delays or prevents conduction of another Whether interference occurs in the sinoatrial junction atria ventricles or as described below in the atrioventricular junction it tends to appear under the following circumstances

Rapid discharge of impulses by a single pacemaker —If for example two impulses are discharged by an atrial pacemaker in close succession, the second impulse may arrive at the atrioventricular node before it has recovered from the effects of the first (Fig 232 A) In this event the second atrial beat may be conducted through the atrioventricular node more slowly than the preceding beat it may penetrate without completely traversing the atrioventricular node or it may not be conducted at all The fact that the impulse either fails to be conducted or is conducted slowly is not a reflection of increased or prolonged refractoriness of the atrioventricular junctional tissues it is a *normal physiologic manifestation* called interference and is due to arrival of the impulse during the normal refractory period of the atrioventricular node Atrioventricular interference plays a prominent role in many of the arrhythmias described in later chapters

Simultaneous activity of two pacemakers —For atrioventricular interference to occur in this condition a supranodal pacemaker (e g sinus node) and an atrioventricular nodal or ventricular center must be active simultaneously (Fig 232 B) When a sinus

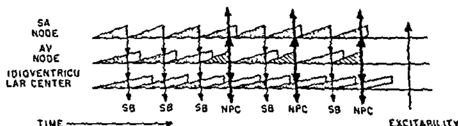


Fig 230 —The mechanism of premature ectopic beats (The conventions here are the same as those in Figures 228 and 229) It can be seen that the sinoatrial node is the dominant pacemaker except on three occasions when an atrioventricular nodal extrasystole or nodal premature contraction (NPC) is discharged early enough to discharge prematurely the sinoatrial node and idioventricular center therefore capturing both the atria and the ventricles Thus the atrioventricular node dominates the higher pacemaker not by virtue of any slowing of the rate of impulse formation in the higher center but rather because of a faster rate of impulse formation in the nodal center itself Any pacemaker outside the sinoatrial node with an inherent rhythmicity so enhanced is called an *ectopic pacemaker* and the beats (or rhythm) it produces are called *ectopic beats* (or an *ectopic rhythm*)

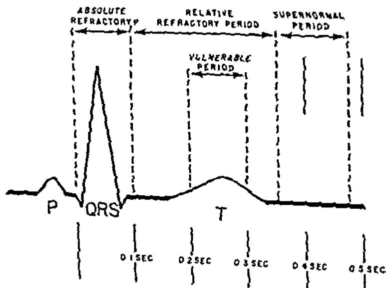


Fig 231 —Absolute refractory period relative refractory period and supernormal period of the atrioventricular and intraventricular conducting pathways and of ventricular muscle shown in relation to the P–QRS–T deflections of the electrocardiogram The vulnerable period cannot be placed in its exact time relationship but is approximately coincident with the peak of the T wave in the electrocardiogram During the vulnerable period, any ectopic impulse which arrives in the ventricles is peculiarly predisposed to set off a series of ectopic beats and to produce an ectopic tachycardia.

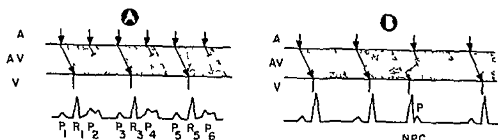


Fig 232 —The two ways in which interference may occur in the atrioventricular junctional tissues On the left of each diagram the letter A indicates the atrial beats or P waves the portion of the diagram enclosed by the two parallel and transverse lines (*AV*) represents the atrioventricular junctional tissues and *V* refers to the ventricular beats Stippled areas correspond to the refractory period of the atrioventricular node following conduction of an impulse In A it is assumed that the sinus node is firing off at a rapid rate and that because of the sinus tachycardia every other impulse arrives at the atrioventricular junction during the normal refractory period following conduction of the preceding sinus impulse Therefore since every other sinus beat fails to be conducted into the ventricles the result is a 2 1 atrioventricular response In B the sinus node is discharging at a relatively slow rate After the second sinus beat an atrioventricu

Consequently since the ventricles respond to the premature atrioventricular nodal extrasystole while the atria are activated by the sinus impulse there results dissociation of the atria and ventricles for this one cycle Atrioventricular interference occurring repeatedly between sinus impulse and impulses arising either in the atrioventricular node or in a lower center is called *atrioventricular dissociation*

impulse and an atrioventricular nodal impulse are fired off almost synchronously each impulse travels toward the other and leaves in its wake refractory muscle When eventually the two impulses meet somewhere in the atrioventricular junctional tissues their further spread is prevented by the refractory tissues ahead so that each in effect cancels out or interferes with further propagation of the other In this instance atrioventricular interference results in the atria and ventricles responding independently to two separate pacemakers for a period of one cycle If interference takes place repeatedly between successive sinus and nodal impulses the two pacemakers continue to discharge independently and the atrial and ventricular rhythms become dissociated This phenomenon is known as *atrioventricular dissociation*

Sometimes it happens that the sinus and nodal impulses meet in the atria rather than in the junctional tissues (Fig 233) Before obliterating each other each impulse activates a portion of the atrial myocardium and therefore contributes to the configuration of the resulting atrial beat A P wave having a configuration intermediate between that of a sinus P wave and a retrograde P wave (an inverted P wave in leads II III and aVF which is produced by retrograde activation of the atria by an atrioventricular nodal or ventricular impulse) and appearing at about the expected time of onset of the next sinus beat is known as an *atrial fusion beat* or a *fusion P wave* Its configuration reflects the relative proportions of the atrial musculature activated by the sinus impulse and by the nodal impulse An atrial fusion beat is a manifestation of atrial interference In a similar manner ventricular fusion beats have a configuration intermediate between that of a conducted sinus beat and an ectopic ventricular beat and are produced by fusion of a supraventricular impulse and an impulse arising in an idioventricular focus A ventricular deflection of this type is indicative of the occurrence of ventricular interference Sinoatrial interference is perhaps observed most commonly with atrioventricular nodal beats Thus a premature nodal impulse may activate the atria and produce a retrograde *P* wave but the next sinus P wave appears at the usual time just as if the nodal beat had not occurred From this it can be inferred that the nodal and sinus impulses interfered with each other at or near the sinoatrial junction since the sinus impulse did not reach the atrial myocardium and the ectopic impulse failed to discharge prematurely the sinoatrial node and to interrupt its rhythm

Atrioventricular Block

Atrioventricular interference and dissociation (Fig 234 A) are not to be confused with *atrioventricular block* which is a manifestation of increased refractoriness of the junctional tissues When an atrial impulse which is expected to pass through the atrioventricular node fails to do so it can be assumed usually correctly that the refractory period of the node is prolonged and that atrioventricular block is therefore

present In its various forms atrioventricular block may occur as the single or most significant abnormality in the electrocardiogram or it may appear as a complication of secondary importance during arrhythmias owing to disturbed impulse formation

In terms of severity or degree atrioventricular block may be divided into the following types incomplete atrioventricular block of first and second degree and complete (third-degree) atrioventricular block

INCOMPLETE ATRIOVENTRICULAR BLOCK First degree block —In this type of atrioventricular block each atrial impulse is conducted to the ventricles at a lower rate than normal The P–R intervals of the

the uncommon type and the common type with the Wenckebach phenomenon

In the uncommon type the P–R intervals of the conducted beats remain constant (Fig 234 B) also atrioventricular conduction of the atrial beats may be blocked at regular intervals so that for example every sixth P wave fails to be followed by a QRS complex On the other hand blocking may occur irregularly without a fixed pattern

In the common type with the Wenckebach phenomenon there is progressive lengthening of the P–R intervals of successive conducted beats until finally one is blocked completely in the atrioventricular node (Fig 234 C) After this pause the next atrial beat which is conducted with a shorter P–R interval than subsequent beats initiates another cycle of lengthening P–R intervals Atrioventricular block showing the Wenckebach phenomenon is the common form of second-degree block and is due to progressively less complete recovery of the depressed junctional tissues after each conducted beat Eventually an atrial impulse arrives during the absolute refractory phase and its failure to be conducted permits the atrioventricular node to recover more completely before the next impulse appears

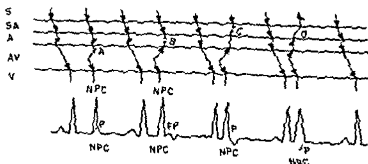


Fig 233 — impulses (Sy... or P waves A premature co... tivation of the follows

A atrioventricular interference The sinus and nodal impulses meet and obliterate each other in the atrioventricular junctional tissues (first NPC on the left)

B atrial interference When interference occurs in the atria between a sinus impulse and a retrograde atrioventricular nodal or ventricular impulse an atrial fusion beat (FP) results since the atria are activated in part by each impulse (second NPC followed by a diphasic P wave)

C sinoatrial interference In this instance the site at which interference takes place between the sinus and retro-

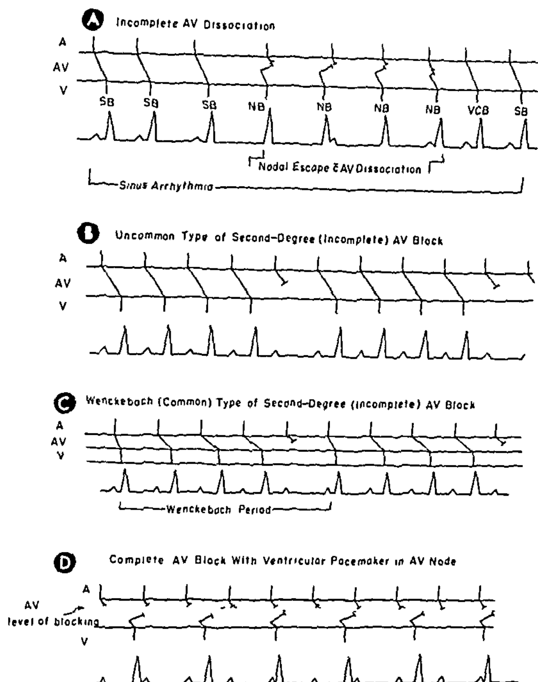


Fig 234 —Mechanisms of atrioventricular nodal escape with atrioventricular dissociation and of incomplete and complete atrioventricular block In **A** *sinus arrhythmia with marked variation in the sinus cycle length* is demonstrated the sinus beats being designated *SB* At one point the sinus pacemaker slows to such an extent that the atrioventricular node is able to escape for four cycles of the ventricular rhythm the nodal beat being labeled *NB* Atrioventricular interference occurs between the retrograde atrioventricular nodal impulses and the descending sinus impulses and so the atrial P waves during this period are of sinus origin The second to the last ventricular deflection is a ventricular capture beat (*VCB*) Such a beat occurs in atrioventricular dissociation whenever a sinus impulse is able to discharge prematurely the atrioventricular node and to be conducted into the ventricle to elicit a ventricular beat In **B** in contrast to atrioventricular interference atrioventricular block entails an abnormal prolongation of the refractory period of the atrioventricular junctional tissues There is prolonged atrioventricular conduction of the conducted sinus beats (first degree atrioventricular block) while every fifth sinus beat fails completely to emerge from the atrioventricular junctional tissues into the ventricles This diagram represents a rather uncommon type of second degree or incomplete atrioventricular block In **C** the more frequently observed *Wenckebach type* of incomplete atrioventricular block is demonstrated The lengths of the R–R cycles progressively shorten until the pause due to complete blocking of a sinus beat occurs constituting a Wenckebach period Characteristically in this type of block the P–R interval of the first conducted beat following the pause is shorter than that of any subsequent beats Also there is a progressive increase in the

node (Fig 234 D) The ventricular rhythm is produced by a pacemaker located below the site of the block The atria and ventricles therefore beat independently the former usually at a more rapid rate

(See Chapter 27 for more detailed discussion of atrioventricular block.)

The varieties of atrioventricular block just described with reference to antegrade atrioventricular conduction may also involve retrograde atrioventricular conduction. Discussion of retrograde atrioventricular block will be deferred until later (Chapter 26) Most disturbances of sinoatrial conduction are for all intents and purposes impossible to recognize with the exception of incomplete (second-degree) sinoatrial block and even this may be difficult to diagnose

Autonomic Nervous Tone

Impulse formation and conduction in the normal heart are affected by a variety of stimulating and depressing influences which are transmitted to the cardiac tissues by the autonomic nervous system and by the circulating blood The hormones mineral ions and other constituents of the blood bear such a complicated and poorly defined relationship to the cardiac arrhythmias that, for the most part they will not be considered in this review

As will become evident, the fact that the vagus and sympathetic nerves maintain the sinoatrial and atrioventricular junctional tissues under a constant state of nervous tone is of fundamental importance in the genesis of many cardiac arrhythmias The balance between sympathetic stimulation and vagal depression tends to fluctuate more or less continuously as nervous impulses originating elsewhere in the body augment or antagonize sympathetic or vagal

or decrease in vagal tone

The right vagus is distributed mainly to the sinoatrial node and junctional tissues while the left vagus

of vagal stimulation and increased vagal ... are dealt with later in more detail (see Chapter 28 for action of digitalis) include depression both of sinus and atrioventricular node rhythmicity and of sinoatrial and atrioventricular conductivity and prolongation of the refractory period of the junctional tissues

Classification of Disturbances of Impulse Formation

The authors of text utilize the following classification of disturbances of impulse formation

A Normal sinus rhythm

B

2 Sinus arrhythmia

C Escape beats and rhythms arising outside the sinus node because of depressed formation or blocked conduction of impulses originating in a higher pacemaker (as in sinus bradycardia wandering supraventricular pacemaker atrial extrasystoles sinoatrial block and atrioventricular block)

1 Atrioventricular nodal escape beats
2 Persistent atrioventricular nodal rhythms with complete capture of the atria
3 Transient atrioventricular nodal rhythms with intermittent atrial interference or incomplete capture of the atria
4 Atrioventricular nodal rhythms with atrioventricular dissociation (without atrial capture)
5 Ventricular escape beats
6 Idioventricular rhythm

D 1

1 Coupled ectopic beats (premature beats or extrasystoles) originating in an ectopic atrial nodal or ventricular focus and requiring a trigger impulse
2 Automatic ectopic beats and rhythms arising in a protected center capable of automatic independent impulse formation (parasystole)
3 Paroxysmal ectopic tachycardia of supraventricular (atrial or nodal) or ventricular origin
4 Flutter and fibrillation of the atria or ventricles

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length of the P–R interval of each conducted beat but the maximal increment in the P–R interval occurs between the first and second conducted beats after the pause while the increment in the P–R interval becomes shorter and shorter with each subsequent beat. The Wenckebach period then terminates with complete blocking of one sinus beat As shown in **D** in complete atrioventricular block none of the atrial beats are conducted into the ventricles Consequently the ventricular rhythm is produced by a secondary pacemaking center below the level of the atrioventricular block The ventricular pacemaker is depicted as if situated low in the atrioventricular junctional tissues Although the atria and ventricles may beat independently in complete atrioventricular dissociation as well as in complete atrioventricular block the two mechanisms can usually be differentiated by the relative rates of the atrial and ventricular rhythms Thus in complete atrioventricular block the atrial rhythm is more rapid than the ventricular while the converse generally holds true in the case of complete atrioventricular dissociation

NORMAL SINUS (SINOATRIAL) RHYTHM

Any cardiac rhythm arising in the sinoatrial node is a sinus rhythm but a *normal sinus* or *sinoatrial rhythm* is differentiated from other rhythms, including variant types of sinus rhythm by one or more of the following characteristics

1 Each ventricular complex is preceded by a P wave which is upright in leads I and II and inverted in lead aVR
2 The P–R interval of a conducted sinus beat is 0 12 second or longer
3 The P and QRS deflections appear at a rate of 60–100 per minute (corresponding to a cycle length of 1 00–0 60 second) in adults
4 The sinus rhythm may be slightly irregular but the longest and shortest P–P cycles (or R–R cycles) differ by less than 0 12 second

Sinus rhythm failing to meet the latter two criteria are discussed below

VARIANT TYPES OF SINUS RHYTHM

Sinus Tachycardia and Sinus Bradycardia

In adults a sinus rhythm having a rate faster than 100 beats per minute is called *sinus tachycardia* Although heart rates as fast as 230 beats per minute may be seen in children with sinus tachycardia adults seldom exhibit rates of over 150 beats per minute The few exceptions (i e sinus tachycardias with rates approaching 180 beats per minute) may be mistakenly diagnosed as paroxysmal atrial tachycardias but the tachycardia can usually be identified as one of sinus node origin by the following characteristics

1 The P–P intervals of a sinus tachycardia tend to vary in length particularly when there are spontaneous or induced fluctuations in sympathetic and/or vagal tone but the P–P cycles in paroxysmal atrial tachycardia remain strikingly constant
2 The P waves during sinus tachycardia generally resemble those present during slower sinus rhythm although there may be some increase in their amplitude for reasons previously cited in the discussion of exercise tachycardia (see Chapter 22) If identifiable the P waves during paroxysmal atrial tachycardia often differ from the sinus P waves in configuration but sometimes the difference may be too equivocal to use as a point of distinction

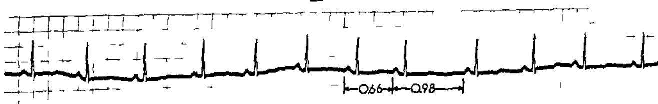
A sinus rhythm with a rate less than 60 (but usually above 45) beats per minute is designated *sinus bradycardia* the necessary assumption being that the atrial rate faithfully indicates the rate of sinus node discharge Occasionally slow sinus rhythms diagnosed as sinus bradycardia may actually be the result of 2 1 sinoatrial block The correct identification of the mechanism is possible only if the onset and offset of the slow rhythm are observed to occur as an abrupt halving or doubling respectively of the atrial rate On the other hand slight irregularity of the slow rhythm favors the diagnosis of sinus bradycardia

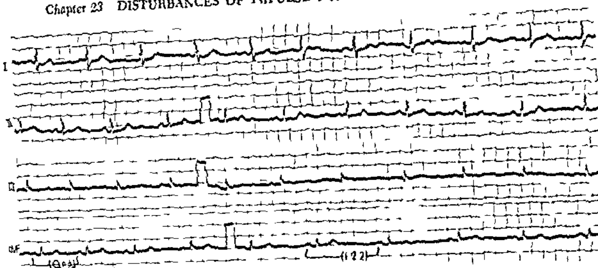
Sinus bradycardia is probably due to a high degree of vagal tone which is thought to displace the cardiac pacemaker to the tail of the sinoatrial node Any influence tending to reduce vagotonia such as exercise excitement and atropine speeds up the atrial rate Sinus bradycardia is observed in many young adults particularly in athletes but is perhaps more commonly seen in older persons with arteriosclerosis

Sinus Arrhythmia

Normal sinus rhythm is ordinarily slightly irregular because of minor physiologic fluctuations in vagal tone When this irregularity is such that the longest

Fig 235 –Sinus arrhythmia in a 10 year-old child with rheumatic fever The diagnosis of sinus arrhythmia can be made in lead II because the difference between the longest and the shortest P–P cycle exceeds 0 12 scond Note that despite the changing sinus cycle length the configuration of the P waves does not change nor do the P–R intervals vary





cycles are indicated in lead aVF. The dif
Sinus arrhythmia is considered a normal

rhythm and has no progno...

and shortest P–P or R–R intervals differ by 0.12 second or more, *sinus arrhythmia* is said to be present (Figs. 235 and 236). It may appear in two forms.

Phasic sinus arrhythmia.—This form of arrhythmia is characterized by an alternate hastening and slowing of the heart rate which coincides with inspiration and expiration. The cyclic stimulation and depression of sinoatrial node rhythmicity are caused by fluctuations in vagal tone as reflex mechanisms in the lungs and blood vessels are activated during expiration and subside with inspiration. Presumably, vagal stimulation suppresses the head of the sinoatrial node so that the pacemaker next in order of rhythmicity (usually the tail of the sinus node) "escapes" and produces a slower rhythm. Subsequent lessening of vagal tone permits the higher center to take over once again.

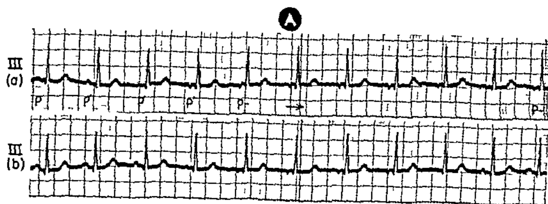
Nonphasic sinus arrhythmia.—In this rhythm, the periodic acceleration and deceleration of the heart is not related to the respiratory cycle. However, this type of sinus arrhythmia may sometimes be converted into the phasic form by forced deep breathing.

The P waves in sinus arrhythmia may become taller and the P–R intervals slightly shorter as the heart rate accelerates, while lower P waves and somewhat longer P–R intervals may accompany slowing of the heart. P–R intervals of less than 0.12 second duration and inverted P waves in leads II, III, and aVF, which characterize beats originating in the atrioventricular node, are not observed in sinus arrhythmia as strictly defined, since migration of the pacemaker is limited to the confines of the sinoatrial node. When, as often happens, atrioventricular nodal beats accompany an otherwise typical sinus arrhythmia, the diagnosis of *wandering supraventricular pacemaker* (see p. 373) is made (Fig. 237, A and B).

Phasic sinus arrhythmia is noted commonly in normal persons, particularly in children, adolescents, and some elderly adults. In contrast, nonphasic sinus arrhythmia is relatively uncommon and tends to occur mainly in older persons with, and sometimes without, heart disease. Occasionally the arrhythmia may be produced or accentuated by digitalis or other agents which increase parasympathetic nervous tone. However, neither form of sinus arrhythmia can be correlated with the presence or absence of cardiac disease.

Coronary Nodal Rhythm

Katz and Pick have applied the term "coronary nodal rhythm" to the electrocardiographic findings of a P–R interval of 0.02–0.10 second and upright P waves in leads I and II (in the absence of QRS changes indicating ventricular pre-excitation). Like Scherf, we do not believe that these findings reflect origin of the rhythm in the region of the coronary sinus but consider the rhythm to be sinoatrial, with the short P–R interval merely representing a normal variation. When the cardiac pacemaker lies in the region of the coronary sinus, the characteristic electrocardiographic findings consist of inverted P waves in leads II, III, and aVF, upright P waves in lead aVR, and a P–R interval longer than 0.12 second. (See p. 366.)



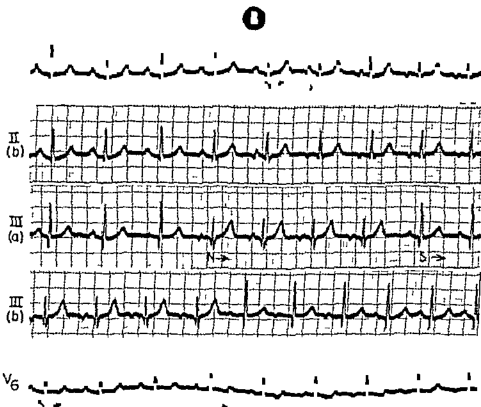


Fig 237 —Two examples of wandering supraventricular pacemaker In A two interrupted strips of lead III are shown Note that the first and second P waves in the top strip lead III (*a*) are upright although differing in appearance while the third P wave is virtually isoelectric The first P wave is a sinus beat (*P*) and the second to fourth P waves are atrial fusion beats (*P*) The ventricular complexes after the first QRS deflection are of atrioventricular nodal origin Subsequently the mechanism of the rhythm is atrioventricular nodal with a retrograde P wave (*P*—) pre

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dicative of first degree atrioventricular block as is also suggested by the prolonged P–R intervals of the sinus beats elsewhere in the lead strips The remaining leads demonstrate features already described where the symbol *S* signifies sinus beats

Atrioventricular Nodal and Idioventricular Escape Beats and Rhythms

ESCAPE BEATS or rhythms appear when impulse formation in a higher center is suppressed or when conduction of the impulses through the heart is blocked. Thus prolonged sinus node inhibition or sinoatrial or atrioventricular block frequently leads to the appearance of escape beats and rhythms arising in the atrioventricular node and less frequently in the ventricles

THE ECG FEATURES OF ATRIOVENTRICULAR NODAL BEATS

Whether atrioventricular nodal beats appear as escape beats or as premature ectopic beats their electrocardiographic features (other than their effect on the cardiac rhythm and time of onset in the cardiac cycle) are essentially the same and can be summarized as follows

1 An excitation impulse discharged by the atrioventricular node is distributed to the ventricles over the usual conducting pathways and therefore produces a QRS deflection resembling the QRS deflections present during sinus rhythm Occasionally the nodal beat may exhibit slight to moderate alterations in QRS configuration and duration (ventricular aberration) as will be described later in this chapter

2 P waves may precede follow or coincide with the ventricular deflections of nodal beats They result from retrograde atrioventricular conduction of the nodal impulses into the atria which are then depolarized in a direction the reverse of that in sinus rhythm Thus the frontal plane mean P vector which is oriented approximately along the +60 axis in normal sinus rhythm is rotated vertically upward

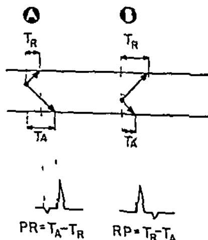
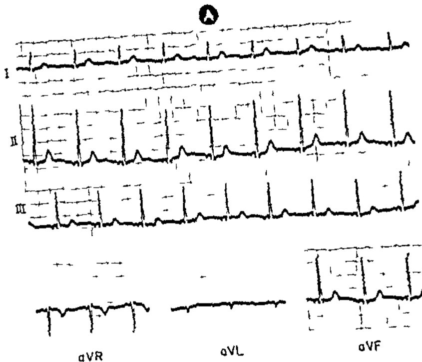


Fig 238 –In A the site of impulse formation is located relatively high in the atrioventricular node and for this reason, forward conduction (T) of the nodal impulse into the ventricles takes a longer period of time than retrograde conduction (T) into the atria Therefore the retrograde P wave precedes the nodal ventricular beat by a P–R interval

[illegible]

uction minus the time required for forward or antegrade conduction of the nodal impulse

Fig 237 —Two examples of wandering supraventricular pacemaker In **A** two interrupted strips of lead III are shown Note that the first and second P waves in the top strip lead III (*a*) are upright although differing in appearance while the third P wave is virtually isoelectric The first P wave is a sinus beat (*P*) and the second to fourth P waves are atrial fusion beats (*P*) *The ventricular complexes after the first QRS deflection are of atrioventricular* nodal origin Subsequently the mechanism of the rhythm is atrioventricular nodal with a retrograde P wave (*P*—) preceding each atrioventricular nodal ventricular beat In lead strip III (*b*) sinus rhythm is present at first then with onset of atrioventricular nodal rhythm atrial fusion P waves appear followed by retrograde P waves The last two complexes in the lower strip are of sinus node origin In **B** as in **A** the P waves labeled *P* are atrial fusion beats those labeled *P* — are retrograde P waves *In lead strip II (a) the first nodal ventricular beat is indicated by* V *and all subse*quent beats in this strip are of atrio the first three
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atrioventricular nodal rhythm and er finding is in
dicative of first degree atrioventricular block as is also suggested by the prolonged P–R intervals of the sinus beats elsewhere *in the lead strips The remaining leads demonstrate features already described* where the symbol S signifies sinus beats



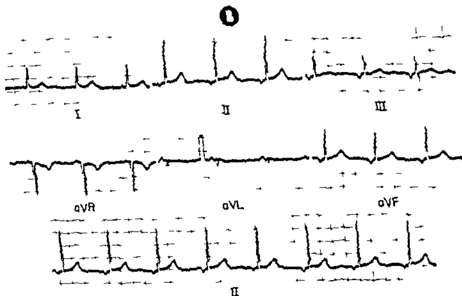
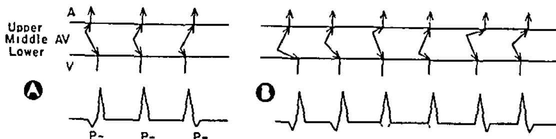


Fig 240 – A and B electrocardiograms obtained from different persons but both demonstrating the typical features of an upper atrioventricular nodal rhythm—namely inverted retrograde P waves in leads II III, and aVF and upright P waves in lead aVR which precede the ventricular complexes by a very short P–R interval The ventricular rate in A is 80 beats per minute



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The upper atrioventric
y a P–R interval of 0 12
d on the ventricular deflection and is obscured In the lower atrioventricular nodal beat the retrograde P wave follows the ventricular beat by an R–P interval of 0 20 second or less B the manner in which the relative rates of antegrade and retrograde conduction of an atrioventricular nodal impulse influence the configuration of the resulting nodal beat The first two electrocardiographic P–QRS deflections are of the type commonly thought to originate in the upper atrioventricular node However the first *atrioventricular nodal beat actually originates low in the* atrioventricular node but antegrade conduction of the impulse is prolonged The second atrioventricular nodal beat originates in the middle of the atrioventricular node but
QRS
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seen that the nodal impulse in one instance originates high in the node while in the other it originates low in the node but in both cases the same period of time is required for antegrade as for retrograde conduction of the atrioventricular nodal impulse In the last two electrocardiographic deflections inverted P waves immediately follow the ventricular deflections This finding is usually ascr
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toward the −90 axis by the retrograde spread of excitation through the atria The vertically superior orientation of s ÂP in retrograde atrial activation is responsible for the fact that the P waves of nodal beats are inverted in leads II III and aVF and upright in lead aVR P waves of the type described are hereafter referred to as *retrograde P waves* and may be produced by impulses arising in lower portions of the atria in the atrioventricular node or in the ventricles

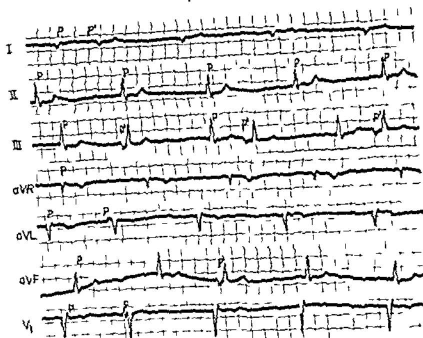
3 The retrograde P wave precedes the QRS complex of an *atrioventricular nodal beat by a P–R* interval of 0 12 second or less or as the case may be follows it by an R–P interval of 0 10–0 20 second

4 *Atrioventricular nodal ectopic and escape beats* can be differentiated electrocardiographically by the fact that onset of an ectopic beat occurs prematurely with respect to the basic cycle length of the *sinus* rhythm while nodal escape beats have a delayed onset

In atrioventricular nodal beats the time relationship between onset of the retrograde P wave and *onset of the QRS complex is* thought to correlate roughly with the level of the pacemaker within the atrioventricular junctional tissues (Figs 238 and 239 A) With this as a basis for differentiation atrioventricular nodal beats may be divided into the following types

Upper atrioventricular nodal beats—An impulse originating in the upper atrioventricular node has only a short distance to spread in a retrograde direction to reach the atria but it must pass through almost the entire length of the node to reach the ventricles As a result retrograde conduction of the nodal impulse is completed more quickly than antegrade conduction and the retrograde P wave of the nodal beat therefore precedes the QRS complex (Figs 240 and 241)

Impulses arising in the atria near the orifice of the coronary sinus vein produce beats which resemble those of upper nodal origin and are called *coronary sinus beats* Because of the caudal site of their origin in the atria coronary sinus impulses activate the atria in a retrograde direction to produce inverted P waves in leads II III and aVF Although these impulses enter the atria directly they must traverse the entire length of the atrioventricular node to reach the ventricles Consequently the retrograde P wave of a



impulse conduction through the atrioventricular node is the same in both antegrade and retrograde directions. However, this is not always the case. As a generalization, it may be said that impaired retrograde conduction of a nodal impulse tends to shift the P wave behind the QRS complex, while disturbed antegrade conduction tends to shift the QRS complex behind the P wave (Fig. 239 B). Thus an apparent lower nodal beat may actually be the result of prolonged retrograde conduction of an upper nodal impulse. Conversely, the appearance of an upper nodal beat may be simulated by prolonged antegrade conduction of a lower nodal impulse. In summary, the P–QRS relationship in atrioventricular nodal beats is determined by two factors: (*a*) the site of impulse origin within the atrioventricular junctional tissues and (*b*) the speed with which the atrioventricular tissues conduct the nodal impulse in a retrograde direction as compared to the rate of conduction in an antegrade direction (Figs. 242 and 243).

ATRIOVENTRICULAR NODAL ESCAPE

It will be recalled that normally the sinus pacemaker keeps the atrioventricular node in a state of suppression owing to the fact that each successive sinus impulse discharges the node prematurely—that is, before the immature impulse forming there can be fired off spontaneously. Since the rhythmicity of the atrioventricular node or of any pacemaker is depressed by premature discharge, which in turn favors con

coronary sinus beat appears relatively sooner and the QRS complex much later after discharge of the excitation impulse than is the case with upper nodal beats The major distinction between the two is that coronary sinus beats have P–R intervals longer than 0 12 second and nodal beats P–R intervals of 0 12 second or shorter However this point of differentiation is far from absolute since upper atrioventricular nodal beats with prolonged forward conduction also exhibit P–R intervals in excess of 0 12 second In any case coronary sinus and upper atrioventricular nodal rhythms probably have the same clinical significance

Middle atrioventricular nodal beats –Antegrade and retrograde conduction of a midnodal impulse require essentially the same amount of time and so activation commences simultaneously in atrial and ventricular myocardium Consequently the characteristic feature of a middle nodal beat is that its retrograde P wave is buried in the larger QRS complex and usually is not discernible

Lower atrioventricular nodal beats –In the case of an impulse arising lower in the atrioventricular node the antegrade atrioventricular pathway is shorter than the retrograde pathway and so ventricular excitation precedes that of the atrial myocardium Ordinarily the ventricular complex of a lower nodal beat is inscribed 0 10–0 20 second earlier than the retrograde P wave provided there is no delay in retrograde conduction

Inasmuch as atrioventricular nodal impulses pass toward the atria and ventricles simultaneously rather than sequentially the P–R or R–P interval of a that classification of atrioventricular nodal beats in the manner just described is valid only if the rate of

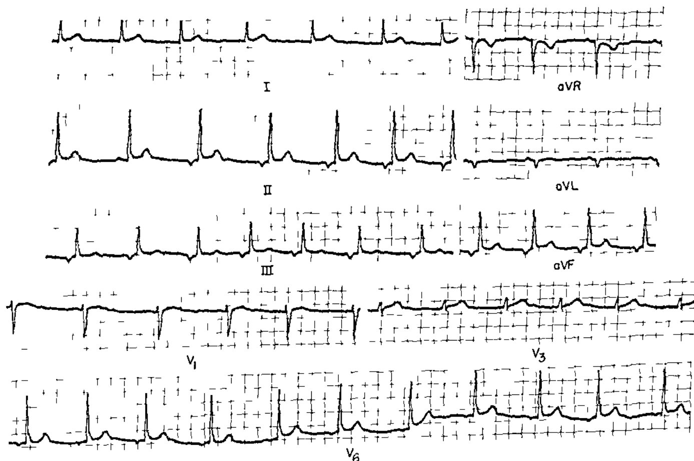


Fig 241 –Electrocardiogram displaying a typical upper atrioventricular nodal rhythm in which the nodal pacemaker drives both the atria and the ventricles The inverted retrograde P waves in leads II III and aVF and the upright P waves in lead aVR precede the ventricular complexes by a P–R interval of 0 13–0 14 second This is slightly longer than the P–R interval usually observed in upper atrioventricular nodal rhythms As an alternative interpretation the electrocardiographic findings could with equal validity represent a coronary sinus rhythm Note in lead II that there is definite arrhythmia of the atrioventricular nodal pacemaker and that one conducted sinus beat appears during the time when the atrioventricular nodal cycle length has lengthened

tionships usually hold true in the electrocardiogram

1 The interval between the last sinus beat and the nodal escape beat exceeds the basic cycle length of the sinus pacemaker (the average P–P interval) and corresponds to the cycle length of the nodal pacemaker

2 In a given lead the intervals preceding each of several nodal escape beats are essentially equal in length This presupposes a constant rate of impulse formation in the atrioventricular node which is usu ... s the case Some variation in the ... tricular nodal arrhythmia the nodal counterpart of sinus arrhythmia

3 Ordinarily the nodal escape beat fails to reach the atria and is not accompanied by a retrograde P wave The reason for this is that in nodal escape the rates of the sinus and nodal pacemakers are usually so nearly the same that their impulses meet and cancel out each other in the atrioventricular junctional tissues (atrioventricular interference) The nodal beat either obscures a superimposed sinus P wave or ... preceded by a nonconducted beat

ATRIOVENTRICULAR NODAL ESCAPE RHYTHMS

When an atrioventricular nodal pacemaker escapes from the sinus node it promptly takes over the ventricular rhythm However in the atria the outcome is more variable in that the node may capture the atria completely incompletely or not at all Since the course of events in the atria following nodal escape may show this variation the electrocardiographic features presented by nodal rhythms may also differ This practical point of distinction makes it possible to arrange nodal rhythms into three overlapping cate-

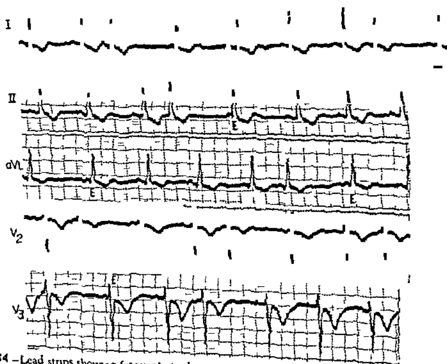


Fig 244 –Lead strips showing frequent atrial n ... h

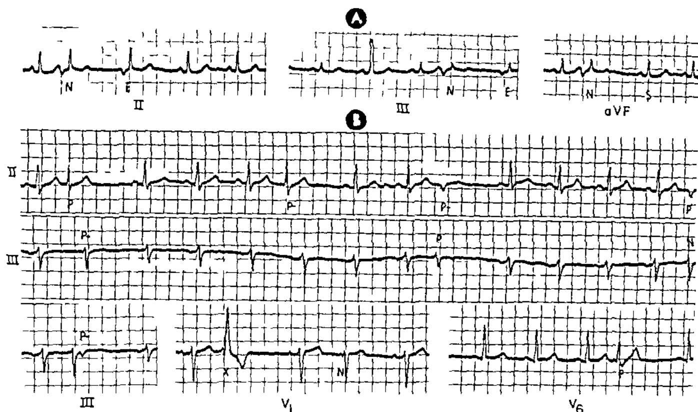


Fig. 243.—Electrocardiographic lead strips showing premature atrioventricular nodal beats of various types. In the lead strips of leads II, III and aVF in A the nodal extrasystoles (N) are preceded by inverted P waves, indicating in all probability an upper atrioventricular nodal origin of the premature beat. In leads II and III the first beat following the premature extrasystole is a nodal escape beat (*E*) which is preceded by a retrograde P wave. This is somewhat unusual in that nodal escape beats ordinarily appear at about the same time as the nonconducted sinus P waves, so that retrograde spread of the nodal impulse into the atria cannot occur. The symbol *S* denotes a conducted sinus beat. Leads II, III, V_1 and V_6 in B were all obtained from the same patient and all contain premature supraventricular extrasystoles. The first two premature beats in lead II and the premature beats in the long strip of lead III show what appear to be terminal S waves but represent in reality fusion of S waves with retrograde inverted P waves (*P−*). Later in lead II inverted P waves of atrioventricular nodal origin can be seen, and these are not accompanied by ventricular beats because of retrograde block of the nodal impulses. In lead III in the lower left corner a typical lower atrioventricular nodal extrasystole is seen, the retrograde P wave following the nodal ventricular beat by an R–P interval of 0.14 second. In the adjacent strip of lead V_1 the extrasystole (X) is probably of ventricular origin. Shortly after there appears an atrioventricular nodal extrasystole with a markedly prolonged coupling interval, preceded by a sinus P wave. In this instance interference between the atrioventricular nodal impulse and the sinus impulse probably occurs in the atrioventricular junctional tissues.

tinued predominance of the sinus node, the suppressing effect of the latter is in a sense self-perpetuating. However, if it should so happen that a sinus impulse fails to reach the atrioventricular node in time to interrupt impulse formation, the nodal pacemaker discharges spontaneously and a nodal escape beat appears. Some of the circumstances in which nodal escape beats may be encountered are listed below.

1. One or more atrioventricular nodal escape beats often terminate a prolonged period of sinus node arrest or pause. Frequently premature discharge of the sinoatrial node by an ectopic atrial beat is followed by a prolonged interval of sinus node inhibition during which one or more nodal escape beats are written (Figs. 244 and 245).

2. Escape beats or a transient nodal rhythm may appear during sinus arrhythmia if the rate of the sinus pacemaker momentarily falls below the inherent rate of impulse formation in the atrioventricular node. This leads to the appearance of a rhythm called *wandering pacemaker* (Fig. 237).

3. Nodal rhythms often emerge during prolonged periods of blocked impulse conduction at the sinoatrial or atrioventricular junction. When the block is of relatively short duration, one or more nodal escape beats may appear before conduction is restored.

When nodal escape beats occur because of delayed arrival of a sinus impulse, the following rela

gories (1) persistent atrioventricular nodal rhythms with [illegible] atrial capture (2) transient or intermittent [illegible] incomplete [illegible] and (3) atrioventricular nodal rhythms [illegible] capture (with atrioventricular dissociation)

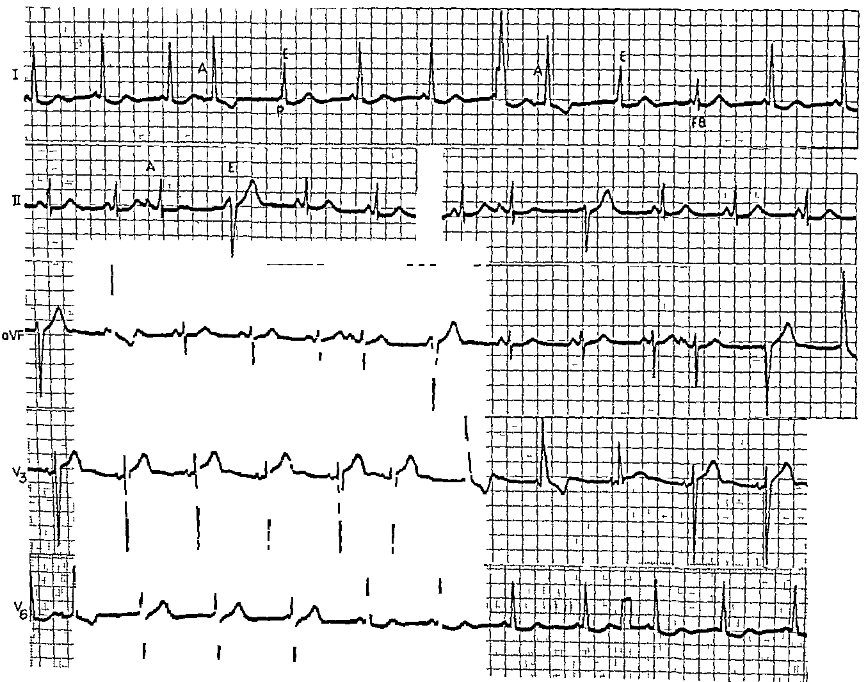
Persisting Atrioventricular Nodal Rhythm with Complete Atrial Capture

Nodal rhythms associated with complete sinoatrial block are included in this category but their discussion will be deferred until later (Chapter 27) along with discussion of the associated conduction disturbances. With these exceptions slow nodal rhythms which pers[illegible] can generally be ascribe[illegible] pression o[illegible] inhibition is secondary to marked vagotonia [illegible] frequently perhaps toxic agents such as excess digitalis quinidine and potassium or inflammatory or degenerative processes are culpable

As previously explained the electrocardiographic appearance of nodal beats depends on the site of the pacemaker in the junctional tissues and on the relative rates of retrograde and antegrade atrioventricular conduction If the retrograde P wave precedes the ventricular deflection the rhythm is said to be an *upper atrioventricular nodal rhythm* if the P wave follows the QRS complex a *lower atrioventricular nodal rhythm* is present and finally nodal ventricular beats unaccompanied by retrograde P waves are indicative of a *middle atrioventricular nodal rhythm* Because of its lower inherent rhythmicity a nodal pacemaker generally drives the heart at a rate of 40–50 beats per minute but unlike the sinus node [illegible] relatively insensitive to variations in nervous tone An atrioventricular nodal rhythm may remain quite regular despite exercise carotid sinus stimulation and similar maneuvers On occasions however [illegible] and is analogous to [illegible] intervals then the diagnosis of *coronary sinus rhythm* can be made

When an atrioventricular nodal focus discharges at a rate of 100 beats per minute or more the resulting rhythm is generally regarded as an ectopic (paroxysmal) atrioventricular nodal tachycardia Presumably the causative disturbance consists of increased excitability of the nodal focus itself with or without concomitant sinus node inhibition Opinions vary as to the proper classification of atrioventricular nodal

Fig 245 — The el[illegible] in Figure 244 displays frequ[illegible] per minute [illegible] (1) Unlike the escape be[illegible] Figure 244 [illegible] ventricular nodal [illegible] originate in the [illegible] QRS interval [illegible] less than 0.12 second [illegible] feature in th[illegible] ventricular fusion beat (IB) and the third beat from the end of lead [illegible] regular fusion beat is generally considered [illegible] ventricular fusion beat [illegible] in which the identity of atrioventricular [illegible] beats was unequivocally established



I
II
aVF
V3
V6
A
E
P
FB

as progressive lengthening of the R–P intervals (Wenckebach phenomenon) reciprocal beats tend to follow the longest R–P interval of each sequence. Thus a ventricular deflection with a normal or prolonged QRS interval (see discussion of ventricular aberration on page 381) which appears prematurely under these circumstances is likely to be a reciprocal beat.

4 The P–R interval of a reciprocal beat can be somewhat prolonged but does not ordinarily exceed the R–P interval. In fact, there seems to be an inverse relationship between the lengths of the R–P interval and the following P–R interval.

5 Generally a reciprocal beat follows the preceding ventricular deflection by an interval of 0.50 second or less.

While it is theoretically possible for reciprocal beats to appear consecutively in the form of a reciprocal rhythm the occurrence of such an arrhythmia in the human heart remains to be proved.

Atrioventricular Nodal Rhythms with Intermittent Atrial Interference (Wandering Supraventricular Pacemaker)

This disturbance is the equivalent of an exaggerated sinus arrhythmia in which vagal depression of the sinoatrial node becomes so marked that the sinus pacemaker fails to keep the atrioventricular node suppressed and the latter escapes for a short period. In keeping with the lower inherent rhythmicity of the atrioventricular node compared to that of the sinus node the nodal beats appear at the relatively slow rate of 40–50 per minute (corresponding to a cycle length of 1.5–1.2 seconds). As in sinus arrhythmia, the supraventricular pacemaker may wander or shift in phase with the respiratory cycle or its movements are nonphasic. In either case there is gradual slowing of the sinus rhythm until the upper atrioventricular node the pacemaker next in descending order of rhythmicity takes over. Occasionally the site of impulse formation may descend even lower in the atrioventricular node. Inasmuch as sinus node depression ordinarily is neither constant in degree nor persistent in duration in this arrhythmia the nodal pacemaker remains active only for sporadic brief periods. With the waning of vagal tone impulse formation in the sinoatrial node gradually speeds up and in the process the sinus node regains its dominance over lower pacemakers. For the most part, the electrocardiographic features of wandering supraventricular pacemaker have been described in previous paragraphs dealing with sinus arrhythmia and nodal escape rhythms and beats. Thus the discussion to follow will center on an additional finding not previously considered in detail namely atrial fusion beats.

Atrial Fusion Beats

During the transition to and from atrioventricular nodal rhythm in wandering supraventricular pacemaker one or more of the nodal beats frequently are preceded by an atrial fusion beat which appears at about the time the next sinus P wave is expected. The configuration of a fusion P wave is a compromise between that of a sinus P wave and of a retrograde P wave but may vary widely within these limits. Atrial fusion beats are produced in the following manner.

1 The nodal impulse passes in a retrograde direction and arrives in the atria at about the same time as the sinus impulse.

2 Thus two coexisting excitation processes are initiated in the atria. One wave of excitation spreads upward from the caudal part of the atria and the other spreads downward from the sinus node.

3 If the two impulses happen to meet in the atria as frequently is the case there occurs a phenomenon known as *atrial interference*. By this is meant that each impulse cancels out the other because both have left refractory muscle in their wake. Thus further spread of either impulse is prevented by the state of refractoriness ahead.

4 However prior to their meeting each impulse has activated a portion of the atrial myocardium. The magnitude of normally directed P wave potentials which are produced will depend on the amount of atrial muscle activated by the sinus impulse. Similarly the more extensive the spread of retrograde excitation through the atria the larger are the resulting retrograde P wave potentials. Variations in the relative times of onset of sinus and retrograde excitation may produce fusion P waves having a configuration intermediate between that of sinus and retrograde P waves or resembling closely one or the other. Thus atrial fusion beats can be upright low diphasic isoelectric or inverted (Fig 237).

As with sinus arrhythmia the occurrence of wandering supraventricular pacemaker is usually secondary to marked vagotonia and is a common finding in normal subjects. It has no significance whatsoever in terms of the presence or absence of underlying cardiac disease.

Fig 246 —Mechanism of reciprocal beats According to Scherf there is longitudinal dissociation of the atrioventricular conducting fibers and so theoretically some of the fibers (*diagonal lines*) may be *completely refractory* and nonresponsive to retrograde spread of an atrioventricular nodal impulse originating at the nodal ectopic focus (N) while others (*stippled*) are *only partially refractory and conduct* the retrograde nodal impulse at a slower rate than normal So slowly is the nodal impulse conducted by the partially refractory atrioventricular fibers that by the time the retrograde impulse reaches *the atria* the previously refractory fibers of the atrioventricular conducting pathway have recovered Consequently as the nodal impulse enters the atria to activate them in a *retrograde direction* it also turns backward and proceeds down the atrioventricular node to traverse the previously refractory atrioventricular fibers in an integrade direction This course of events is reflected in the electro[...] *P wave sand*wiched between two v[...] [...]lection is produced by the original [...] retrograde P wave follows it by a pro[...] [...]ar deflection produced by the re entry impulse is called a *reciprocal beat (RB)* the interval between onset of the initial nodal beat and onset of the reciprocal beat being 0 50 second or less

rhythms with rates above 50 but less than 100 beats per minute It has been suggested that these rhythms be considered the nodal counterpart of sinus tachycardia (nonparoxysmal nodal tachycardia) (*Fig 240*)

Reciprocal Beats in Atrioventricular Nodal Rhythm

In occasional atrioventricular nodal rhythms with initial excitation of the ventricles (and *rarely in association* with premature nodal or ventricular ectopic beats) a retrograde P wave may appear sandwiched between two closely spaced *ventricular* beats The second of these is written prematurely with respect to the usual R–R interval of the prevailing nodal rhythm and is known as a *reciprocal beat* The following explanation has been offered for the genesis of reciprocal beat (Fig 246)

Above the site of impulse formation in the atrioventricular node there is a localized area of depression where some longitudinal fiber pathways are involved *more severely than others* As a result not only is there unidirectional blocking of retrograde impulse conduction in some fibers but there is also delayed retrograde conduction throughout the entire area of refractoriness As the nodal impulse producing the initial ventricular beat spreads through the ventricular myocardium it also travels up the atrioventricular node in a retrograde direction and soon enters the area of depression There the retrograde impulse is completely blocked in the severely depressed fibers but passes slowly up the remaining less refractory atrioventricular pathways Because of this delay in retrograde conduction the retrograde P wave follows the QRS complex of the nodal beat by a prolonged R–P interval (usually 0 20 second or longer) If the R–P interval is sufficiently prolonged the longitudinal atrioventricular fibers which were previously *completely* refractory to the retrograde nodal impulse in the meantime recover their conductivity In this event the retrograde impulse splits before entering the atria turns back and passes down the atrioventricular node in an integrade direction along the now responsive conducting fibers The time required for retrograde conduction of the nodal impulse and subsequent forward conduction of the re entry impulse is sometimes long enough to permit the ventricles to recover after their initial response to the nodal impulse If such is the case the re-entry impulse elicits a second ventricular response or *reciprocal beat* (Fig 249)

The electrocardiographic recognition of reciprocal beats encounters its chief obstacle in differentiating these beats from ventricular capture beats (or interference beats) *which occur in nodal rhythms with* atrioventricular dissociation Both types of beats may appear in similar arrhythmias and in fact even in a single electrocardiogram but ventricular capture beats are by far the more common finding The points summarized below are useful in the identification of reciprocal beats

1 The P wave preceding a reciprocal beat must have the configuration of a fusion P wave or preferably a retrograde P wave Its contour must therefore differ from that of the sinus P wave

2 Reciprocal beats are most likely to occur in nodal rhythms with delayed retrograde conduction as shown by R–P intervals in excess of 0 20 second

3 When the retrograde conduction delay occurs

as progressive lengthening of the R–P intervals (Wenckebach phenomenon) reciprocal beats tend to follow the longest R–P interval of each sequence. Thus a ventricular deflection with a normal or prolonged QRS ... cussion of ventricular aberration on ... under these ... beat.

4 The P–R interval of a reciprocal beat can be somewhat prolonged but does not ordinarily exceed the R–P interval. In fact there seems to be an inverse relationship between the length of the R–P interval and the following P–R interval.

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While it is theoretically possible for reciprocal beats to appear consecutively in the form of a reciprocal rhythm, the occurrence of such an arrhythmia in the human heart remains to be proved.

Atrioventricular Nodal Rhythms with Intermittent Atrial Interference (Wandering Supraventricular Pacemaker)

This disturbance is the equivalent of an exaggerated sinus arrhythmia in which vagal depression of the sinoatrial node becomes so marked that the sinus pacemaker fails to keep the atrioventricular node suppressed and the latter escapes for a short period. In keeping with the lower inherent rhythmicity of the atrioventricular node compared to that of the sinus node, the nodal beats appear at the relatively slow rate of 40–50 per minute (corresponding to a cycle length of 1.5–1.2 seconds). As in sinus arrhythmia, the supraventricular pacemaker may wander or shift in phase with the respiratory cycle, or its movements are nonphasic. In either case there is gradual slowing of the sinus rhythm until the upper atrioventricular node, the pacemaker next in descending order of rhythmicity, takes over. Occasionally the site of impulse formation may descend even lower in the atrioventricular node. Inasmuch as sinus node depression ordinarily is neither constant in degree nor persistent in duration in this arrhythmia, the nodal pacemaker remains active only for sporadic brief periods. With the waning of vagal tone, impulse formation in the sinoatrial node gradually speeds up and in the process the sinus node regains its dominance over lower pacemakers. For the most part the electrocardiographic features of wandering supraventricular pacemaker have been described in previous paragraphs dealing with sinus arrhythmia and nodal escape rhythms and beats. Thus the discussion to follow will center on an additional finding not previously considered in detail, namely, atrial fusion beats.

Atrial Fusion Beats

During the transition to and from atrioventricular nodal rhythm in wandering supraventricular pacemaker, one or more of the nodal beats frequently are preceded by an atrial fusion beat which appears at about the time the next sinus P wave is expected. The configuration of a fusion P wave is a compromise between that of a sinus P wave and of a retrograde P wave but may vary widely within these limits. Atrial fusion beats are produced in the following manner.

1 The nodal impulse passes in a retrograde direction and arrives in the atria at about the same time as the sinus impulse.

2 Thus two coexisting excitation processes are initiated in the atria. One wave of excitation spreads upward from the caudal part of the atria, and the other spreads downward from the sinus node.

3 If the two impulses happen to meet in the atria, as frequently is the case, there occurs a phenomenon known as *atrial interference*. By this is meant that each impulse cancels out the other because both have left refractory muscle in their wake. Thus further spread of either impulse is prevented by the state of refractoriness ahead.

4 However, prior to their meeting, each impulse has activated a portion of the atrial myocardium. The magnitude of normally directed P wave potentials which are produced will depend on the amount of atrial muscle activated by the sinus impulse. Similarly, the more extensive the spread of retrograde excitation through the atria, the larger are the resulting retrograde P wave potentials. Variations in the relative times of onset of sinus and retrograde excitation may produce fusion P waves having a configuration intermediate between that of sinus and retrograde P waves or resembling closely one or the other. Thus atrial fusion beats can be upright, low, diphasic, isoelectric or inverted (Fig. 237).

As with sinus arrhythmia, the occurrence of wandering supraventricular pacemaker is usually secondary to marked vagotonia and is a common finding in normal subjects. It has no significance whatsoever in terms of the presence or absence of underlying cardiac disease.

Atrioventricular Nodal Rhythms without Atrial Capture (with Atrioventricular Dissociation)

Nodal rhythms with atrioventricular dissociation differ from those previously described in that the nodal pacemaker produces only the ventricular rhythm, while the atria remain completely under the control of the sinoatrial node Dissociation of the two pacemakers occurs within the atrioventricular junctional tissues at a level above the nodal center where one of two mechanisms prevents antegrade conduction of sinus impulses and retrograde conduction of nodal impulses These mechanisms are atrioventricular interference and atrioventricular block

Atrioventricular interference —For atrioventricular interference to occur repeatedly as it must in dissociation the timing and rate of the two pacemakers must be such that each sinus impulse reaches the atrioventricular node in time for interference to occur between it and the retrograde nodal impulse

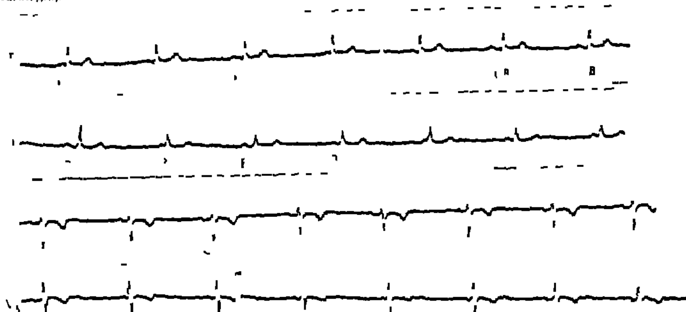
Atrioventricular block —The block may affect both antegrade and retrograde atrioventricular conduction or it may be unidirectional and involve either Dissociation of a faster nodal rhythm and a slower sinus rhythm the common form of persisting atrioventricular dissociation is often accompanied by retrograde atrioventricular block

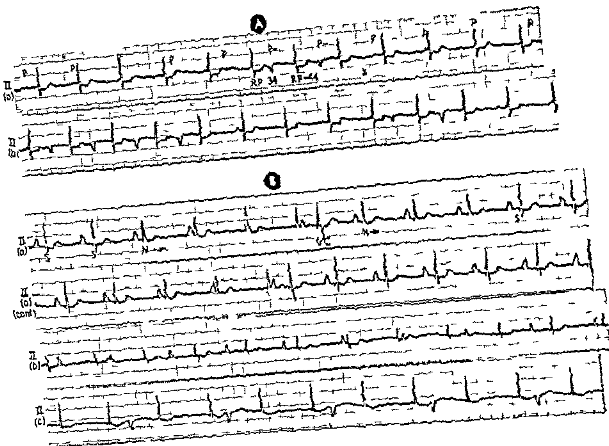
Atrioventricular dissociation may be complete or incomplete *Complete dissociation* is characterized by the fact that not a single sinus or nodal impulse passes completely through the atrioventricular node the atrial and ventricular rhythms remaining absolutely independent of each other *Incomplete dissociation* occurs when an occasional sinus impulse finds the atrioventricular node responsive and is conducted into the ventricles to produce a *ventricular capture beat* By capturing the ventricles for one or more beats the conducted impulse momentarily interrupts the nodal pacemaker Rarely incomplete dissociation is observed in which an occasional nodal impulse is conducted in a retrograde direction and captures the atria

Complete Atrioventricular Dissociation

In the absence of atrioventricular block physiologic interference alone may produce complete atrioventricular dissociation provided the sinus and nodal

Fig 247 —Atrioventricular dissociation This electrocardiogram demonstrates the simplest most common and most benign form of atrioventricular dissociation There is a slow sinoatrial rhythm with eventual atrioventricular nodal escape the nodal pacemaker escaping at its inherent rate of impulse formation to produce a slow nodal rhythm in the ventricles There are occasional ventricular capture beats (*CB*) occurring after slightly shorter R–R cycles than those of the atrioventricular nodal beats In this record the main clue to the presence of atrioventricular dissociation is the varying P–R r
tion The pres
sons (1) th
Q–T interv
and (3) the
cardiograph





ventricular
of lead II s
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pacemakers discharge at essentially the same rate. Rate equalization is accomplished by acceleration of the nodal pacemaker and/or slowing of the sinus node. Ordinarily complete dissociation due to interference is an intermittent and transient phenomenon appearing during sinus bradycardia, sinus arrhythmia or wandering pacemaker (Fig 247). It is observed in a more persistent form in coexisting sinus and nodal tachycardia and more rarely in combined or double paroxysmal tachycardias.

Complete dissociation of a fast sinus rhythm and a lower nodal rhythm cannot be attributed solely to physiologic interference which, as mentioned previously, requires equalization of the two pacemakers' rates. Antegrade atrioventricular block must therefore be implicated. Complete dissociation with antegrade atrioventricular block is described later in more detail; however, as a general rule the degree of atrioventricular block required to produce dissociation increases as the difference in the rates of the sinus and nodal rhythms become greater.

Incomplete Atrioventricular Dissociation

Atrioventricular dissociation which tends to persist for a relatively long time commonly appears as incom

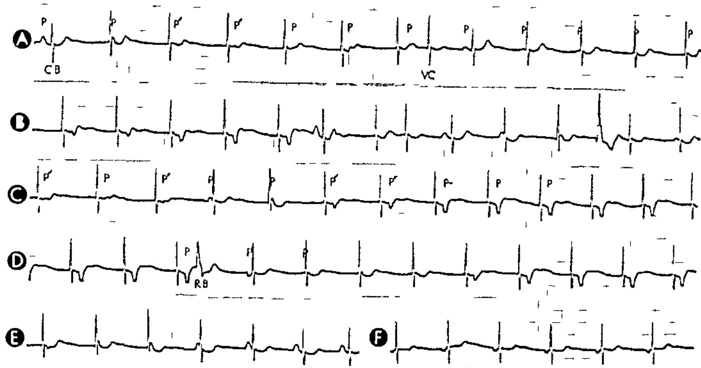


Fig 249 —Atrioventricular nodal rhythm with alternating periods of atrioventricular dissociation and retrograde atrial activation with retrograde atrioventricular block All lead strips are lead II The ventricular complex (*CB*) is a conducted sinus beat the ventricular deflection (*VC*) is a ventricular capture beat and the QRS deflection (*RB*) is a reciprocal beat P waves of sinus node origin are designated *P* those of atrioventricular nodal origin *P*– and those labeled *P* *are atrial fusion beats Note that the configuration of the conducted sinus beat in* **A** *differs from that of atrio*ventricular nodal beats elsewhere in the same lead strip In **D** the retrograde P wave following the third nodal beat is followed in turn by a ventricular deflection showing aberration The interval between the preceding nodal ventricular beat and the ventricular deflection following the retrograde P wave is 0 36 second while the R–P interval of the nodal beat is 0 16 second In lead strips **B** **C** and **D** the alternating appearance of sinus and retrograde P waves suggests that retrograde atrioventricular block must be present at times With the single exception of the relatively short R–P interval of the third nodal beat in **D** all other features of the premature ventricular deflection (*RB*) are compatible with this beat being a reciprocal beat In **E** a mechanism is demonstrated which may tend either to perpetuate atrioventric

ventricular nodal rhythm

plete dissociation of a rapid nodal rhythm and a slower sinus rhythm Incomplete dissociation may also occur intermittently as will be indicated later in the discussion Parenthetically it should be pointed out that many texts apply the term *atrioventricular dissociation* only to the form of incomplete dissociation described above However in this text the term is used to designate a mechanism which occurs with various rhythms although observed most frequently with nodal rhythm

In incomplete dissociation of a faster nodal rhythm and slower sinus rhythm the difference in the rates of the two pacemakers is usually due to an accelerated *rate of impulse formation in the atrioventricular node* although slowing of the sinoatrial node may be a contributory factor Thus depending on the circumstances incomplete dissociation may occur with nodal rhythms whose rates range from 40 to 100 beats per minute and sometimes even higher if paroxysmal nodal tachycardia is present Since in the electrocardiogram the P–P intervals of the slower atrial rhythm are longer than the R–R intervals of the ventricular rhythm successive P waves appear to march into and then emerge behind the QRS complexes However even though the relative positions of the sinus P waves and nodal R waves are constantly changing the P–P intervals and R–R intervals may both be spaced off regularly

The inability of the faster nodal center to take over the atrial rhythm is due to the fact that retrograde *conduction of the nodal impulse is prevented* either by physiologic atrioventricular interference or by retrograde atrioventricular block (Figs 248 and 249) Interference is the mechanism more likely to be in

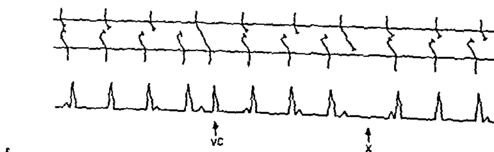
solved when the rates of the sinus node and nodal pacemaker do not *differ* greatly If such proves to be the case two additional factors may tend to favor the interference mechanism (1) Retrograde atrioventricular conduction takes place *less readily and more* slowly than antegrade conduction. This fact may off

do not differ greatly a form of sinus arrhythmia may be observed which resembles ventriculophasic sinus arrhythmia occurring in complete atrioventricular block As mentioned above the P waves usually march into and through the QRS complexes However in the case of sinus arrhythmia the first P wave to emerge behind the ventricular beat appears after a *shorter P–P interval than those preceding its earlier onset being related in some way to ventricular systole* Because of the shortened P–P interval *the P wave* moves back in front of the QRS *complex again* and the cycle repeats itself This mechanism *makes it possible* for the sinus P wave to remain in close proximity to the nodal beat thereby *enabling the sinus impulse* to interfere with the *retrograde nodal impulse repeatedly*

More often than not in fast nodal *rhythms incomplete* dissociation is maintained by a unidirectional blocking of retrograde atrioventricular conduction as evidenced by failure of a retrograde P wave to appear when *expected (A retrograde P wave should follow* within 0 20 second or less the QRS complex of the nodal beat whenever *the next sinus* P wave falls well outside the Q–T *interval of the ventricular beat)*

Since the nodal impulses are *discharged* more rapidly than the sinus impulses can arrive in the junctional tissues most of the sinus impulses find the atrioventricular node refractory following excitation by a previous nodal beat *Nonetheless* because of the different timing of the two pacemakers an occasional sinus impulse arrives at the atrioventricular node outside its absolute refractory phase and therefore is conducted into the ventricles If the conducted impulse finds the ventricles responsive it initiates ventricular excitation prematurely—that is prior to onset of the *expected nodal beat* The resulting ventricular deflection is called a *ventricular capture beat* and ordinarily presents much the same appearance as the nodal beats previously *recorded There is an inverse relationship* between the P–R *interval of the ventricular capture* beat and the preceding R–P interval *When the R–P* interval is short the sinus *impulse arrives earlier in* the relative refractory phase of the atrioventricular node and is *conducted to the ventricles* more slowly than an impulse arriving later after a longer R–P interval Sometimes onset *of the ventricular capture* beat occurs so prematurely that the recovery process in the ventricles is interrupted before its completion *Because of the partial refractoriness* of the ventricular muscle there is aberrant spread of excitation through the ventricles This is manifested in the ventricular *capture beat by the appearance* of slight to marked *changes in* QRS *configuration* and/or duration *Ventricular aberration* is the term used to refer to the QRS *changes resulting from aberrant* intraventricular conduction or from *preferential atrioventricular conduction* (discussed later on page 381)

In passing through the atrioventricular node the conducted *atrial impulse of a ventricular* capture beat



... uar conduction. ... atrioventricular nodal

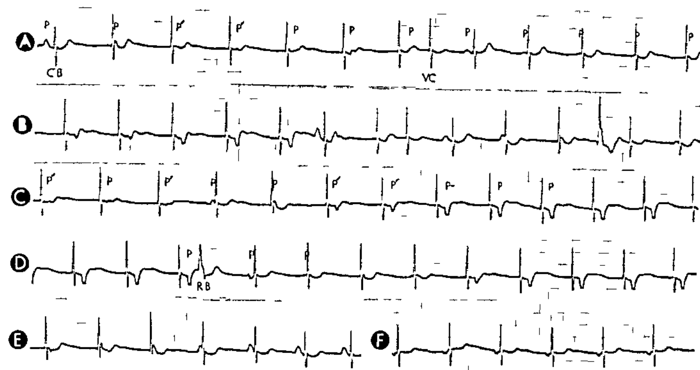


Fig 249 —Atrioventricular nodal rhythm with *alternating periods of atrioventricular dissociation and retrograde* atrial activation with retrograde atrioventricular block All lead strips are lead II The ventricular complex (*CB*) is a conducted sinus beat the ventricular deflection (*VC*) is a ventricular capture beat and the QRS deflection (*RB*) is a reciprocal beat P waves of sinus node origin are designated *P* those of atrioventricular nodal origin *P*— and those labeled *P* are atrial fusion beats Note that the configuration of the conducted sinus beat in **A** differs from that of atrioventricular nodal beats elsewhere in the same lead strip In **D** the retrograde P wave following the third nodal beat is

val of the third nodal beat in **D** all other features of the premature ventricular deflection (*RB*) are compatible with this beat being a reciprocal beat In **E** a mechanism is demonstrated which may tend either to perpetuate atrioventricular dissociation or to restore sinus rhythm This mechanism is a form of ventriculophasic sinus arrhythmia Thus in **E** the sinus P wave can be seen to emerge gradually behind the QRS deflection of the first three nodal beats however the third sinus cycle is shortened causing the sinus P wave to move back in front of the fourth nodal ventricular beat The last two QRS deflections in this lead strip are probably conducted sinus beats In **F** there is a persisting upper atrioventricular nodal rhythm

plete dissociation of a rapid nodal rhythm and a slower sinus rhythm Incomplete dissociation may also occur intermittently as will be indicated later in the discussion Parenthetically it should be pointed out that many texts apply the term *atrioventricular dissociation* only to the form of incomplete dissociation described above However in this text the term is used to designate a mechanism which occurs with various rhythms although observed most frequently with nodal rhythm

In incomplete dissociation of a faster nodal rhythm and slower sinus rhythm the difference in the rates of the two pacemakers is usually due to an accelerated *rate of impulse formation in the atrioventricular node* although slowing of the sinoatrial node may be a contributory factor Thus depending on the circumstances incomplete dissociation may occur with nodal rhythms whose rates range from 40 to 100 beats per minute and sometimes even higher if paroxysmal nodal tachycardia is present Since in the electrocardiogram the P–P intervals of the slower atrial rhythm are longer than the R–R intervals of the ventricular rhythm successive P waves appear to march into and then emerge behind the QRS complexes However even though the relative positions of the sinus P waves and nodal R waves are constantly changing the P–P intervals and R–R intervals may both be spaced off regularly

The inability of the faster nodal center to take over the atrial rhythm is due to the fact that retrograde *conduction of the* nodal impulse is prevented either by physiologic atrioventricular interference or by retrograde atrioventricular block (Figs 248 and 249) Interference is the mechanism more likely to be in

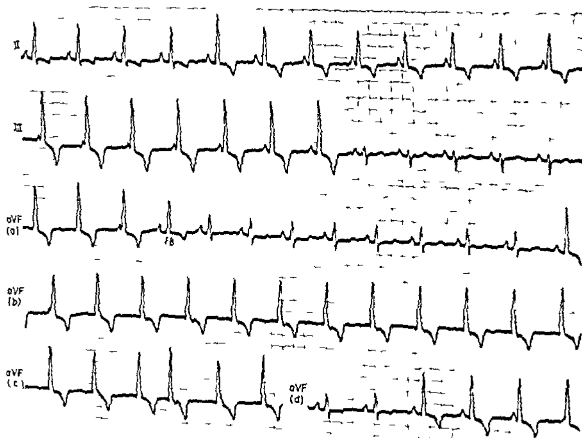
the interval between two consecutive nodal beats the ϵ frequently is a delay in conduction of the sinus impulse through the bundle of His Inasmuch as the nodal pacemaker is discharged *by the sinus impulse* before it reaches the area of conduction delay the next nodal impulse *begins to form well in advance of* onset of ventricular activation and is discharged sooner thereafter Thus the VC–NB interval is short*ened to the extent that ventricular excitation by the conducted atrial impulse is delayed In the authors* experience *shortened VC–NB intervals are observed* quite frequently Occasionally the VC–NB interval is longer than a single-cycle R–R interval of the nodal rhythm. This has been attributed to transient depression of the nodal pacemaker resulting from its premature discharge by the conducted atrial beat (Figs 250 and 251)

Clinical Significance of Atrioventricular Dissociation

[illegible]

cance of this finding Thus [illegible] sinus arrhythmia sinus bradycardia or wandering pacemaker is usually of little consequence while its appearance under different circumstances may be of great diagnostic importance. In short, atrioventricular dissociation derives its clinical significance from the mechanisms and related factors responsible in a given instance for its appearance with reference particularly to the following

1 *Rate of atrioventricular nodal rhythm* – As previously stated *atrioventricular dissociation occurring with slow nodal escape rhythms (rate of 40–50 beats*



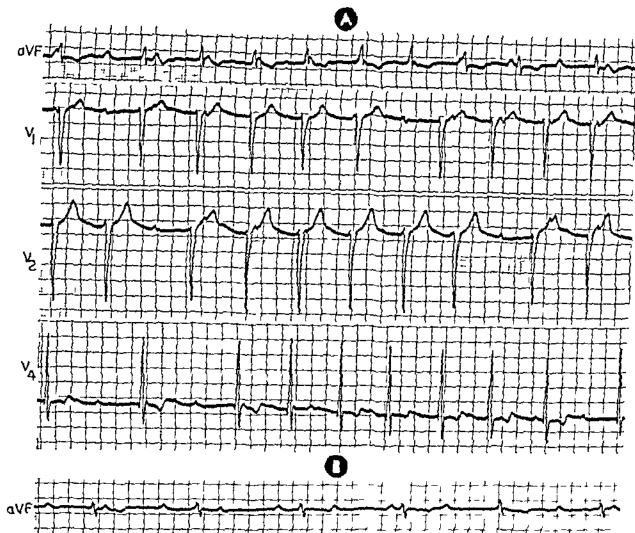


Fig 251 –*In A the electrocardiographic lead strips show for the most part a relatively rapid atrioventricular nodal rhythm with a slower sinus rhythm in the atria There are no definitely recognizable ventricular capture beats However there are frequent pauses in the ventricular rhythm Possibly the best explanation for these pauses is that the sinus P wave which either slightly precedes or is superimposed on the last nodal beat before the pause is conducted almost*
it In so doing it discharges the immature atrioventricular
that the pauses in the ventricular rhythm are the result of
odal beat This explanation is supported somewhat by the
There is complete atrioventricu
deflections resembles that of the
pacemaker in B almost exactly
of the atrioven
re shorter than

discharges the immature impulse forming in the nodal pacemaker If the nodal center *is* then able to reform and discharge a second impulse before arrival of the next sinus impulse at the atrioventricular junction a nodal beat follows the ventricular capture beat However should the sinus impulse arrive first the ventricular capture beat is followed by one or more conducted sinus beats depending on how long the sinus node remains in control of the atria and ventricles When ventricular capture beats initiate short periods of sinus rhythm incomplete dissociation is said to be intermittent rather than sustained

If a ventricular capture beat is followed by a nodal beat the interval between the two (*VC–NB* interval*) sometimes corresponds to a single cycle of the nodal rhythm since it represents the time required by the nodal pacemaker to mature and discharge its next impulse Although the VC–NB interval often equals

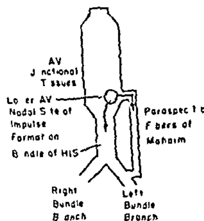
*VC = ventricular capture beat NB = nodal beat

per minute) m
dissociation ac
tachycardia (rate of 90–100 ...
ute) is sometimes the first *indication* of inflammatory or degenerative myocardial disease Later in Chapter ...6 paroxysmal atrioventricular nodal tachycardia (rate above 100 beats per minute) with atrioventricular dissociation will be discussed along with its clinical implications

2 *Mechanism affecting atrioventricular conduction—atrioventricular block versus atrioventricular interference*—Because atrioventricular interference is a

... refractoriness of the intraven...

ventricular muscle This condition ... to marked *intraventr* ...
changes in
are referred ...
Aberration of the ventricular *complex is* ...
obs ... ectopic beats or in ectopic
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...
duction The factor common to all these ...



paraspecific fibers o
function as a conducting pathway ...
the explanation of ventricular aberration of *atrioventricular nodal beats* given here must be considered tentative until further information is available

physiologic phenomenon it does not in itself constitute an abnormality even when interference is the sole *mechanism* responsible for dissociation Failure of ventricular or atrial capture beats to appear when expected establishes the presence of atrioventricular *block* (*antegrade* or retrograde block respectively) which may have the same significance as nodal tachycardia occurring with atrioventricular dissociation

3 *Clinical background*—Atrioventricular dissociation is sometimes produced by inflammatory and degenerative processes involving the myocardium such as rheumatic myocarditis and arteriosclerotic heart disease or by intoxication with certain drugs particularly digitalis and quinidine When dissociation is superimposed on a clinical background of suspected drug intoxication or cardiac disease this finding has greater *significance than* would otherwise be the case

Ventricular Aberration of Atrioventricular Nodal Escape Beats

If an excitation impulse arrives in the ventricles before they have recovered completely from previous

that two or more impulses reach the *ventricles in*

...

that of a right bundle branch block

As indicated previously nodal escape beats are characterized by delayed onset This makes it all the more surprising that minimal to moderate degrees of ventricular aberration are frequently observed in nodal escape beats (Figs 252–254) Obviously the mechanism involved differs from that described for premature beats and rapid rhythms since the late onset of the nodal escape beat would seem to preclude as the causative factor refractoriness of the intraventricular conducting pathways Pick has recently advanced the hypothesis that aberration of nodal escape beats may be the result of preferential atrioventricular conduction of the nodal impulses (Fig. 255) This concept has its basis in the fact that irregular communications of the atrioventricular node common bundle and left bundle branch with the ventricular

Fig 253 —Electrocardiogram showing intermittent incomplete atrioventricular dissociation with atrioventricular nodal rhythm and occasional atrial extrasystoles *However* the most striking feature is the marked difference in the QRS configuration of the sinus beats and the atrioventricular nodal beats Symbols S sinus beats *P* sinus P waves V atrioventricular nodal beats and *FP* atrial fusion beat (occurring in lead aVF)

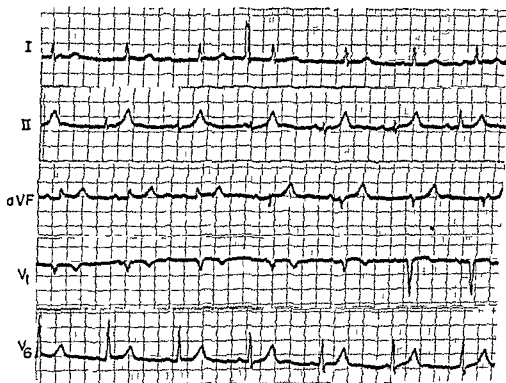


Fig 254 —Intermittent atrioventricular nodal rhythm with incomplete atrioventricular dissociation and ventricular aberration of the nodal beats

Ectopic Beats and Rhythms

TERMINOLOGY AND GENERAL CONSIDERATIONS

ECTOPIC BEAT.—An ectopic beat is a beat which arises outside the sinoatrial node in an ectopic focus and results fr[illegible]
tivity or rhyt[illegible]
origin. An ectopic bea[illegible]
automatic ectopic beat or a coupled ectopic beat.

a) AUTOMATIC ECTOPIC BEATS (PARASYSTOLE).—The focus producing beats of this type is unique in that not only does it form and discharge impulses automatically and independently but its rhythmical activity is "protected" against interruptions by impulses originating in the sinus node or elsewhere. The mechanism giving rise to automatic beats is called *parasystole* and is described later.

Interectopic interval.—This term refers to the interval between two consecutive ectopic beats (see Fig 258) whether or not one or more beats of different origin intervene.

b) COUPLED PREMATURE ECTOPIC BEATS.—In this type of ectopic beat the impulse is not discharged automatically but requires a triggering excitation impulse. The initiating impulse may be a conducted sinus beat or another ectopic beat acting as a trigger impulse or a "re-entry" impulse. Whether the interval [illegible]

Bigeminy.—The term bigeminy is used to describe cardiac rhythms consisting of coupled beats but the mechanism of the paired beats should always be indicated. While bigeminy is usually due to coupled *ectopic beats each of which is initiated by a preceding beat of the prevailing rhythm* it may also be produced by several other mechanisms such as 3:2 Wenckebach atrioventricular block and *nonconducted* atrial extrasystoles following every second sinus beat.

Trigeminy and quadrigeminy.—Trigeminy consists of two coupled ectopic beats following each sinus beat; quadrigeminy of three extrasystoles after each sinus beat. A series of four or more ectopic *beats appearing in rapid succession* may be regarded as a short run of paroxysmal tachycardia.

Multiple coupled ectopic beats.—To indicate the frequency with which extrasystoles occur in a given electrocardiogram the following arbitrary scheme may be used: (*a*) the term *rare extrasystoles* is used if only one ectopic beat occurs in a 10-second interval; (*b*) *the term occasional extrasystoles* signifies the occurrence of two or three extrasystoles in a 10-second interval; and (*c*) the term *frequent extrasystoles* is used when a 10-second interval in the electrocardiogram contains four or more ectopic beats.

Coupling interval.—The coupling interval is the interval between onset of an extrasystole and onset of the beat immediately preceding (see Fig 258). In the case of atrioventricular nodal and ventricular ectopic beats the coupling interval is measured from the QRS complex of the sinus beat to that of the extrasystole, and in atrial premature beats from the sinus P wave to the ectopic P wave. The length of the coupling *interval* is a *measure of the prematurity of* the coupled beat and is determined in part by the *length of the conduction* pathway which must be traversed by the trigger impulse in order to reach the ectopic focus. The length of this pathway and thus the length of the *coupling interval* depends largely on the site of impulse origin. As a corollary to the preceding statement the location of the ectopic focus has an important bearing on the way *the excitation* impulse spreads through the myocardium. The more *abnormal* the spread of excitation the more the ectopic beat differs in appearance from the normal beat

myocardium (variously labeled *paraspecific fibers* or *preferential pathways*) have been demonstrated in normal fetal neonatal and young adult hearts Hypothetically at least it can be postulated that a nodal impulse may sometimes be transmitted via this accessory pathway to some part of the ventricular myocardium instead of utilizing the normal atrioventricular conducting pathway like impulses arising above the atrioventricular node The degree of ventricular aberration displayed by the nodal beat would be related to the length of the preferential pathway and to the point at which excitation is introduced into the ventricular myocardium

Pick has published records showing ventricular aberration of one or several nodal escape beats and examples of this phenomenon appear in figures of this text To these observations we have added a small series of electrocardiograms which show ventricular aberration during relatively sustained rhythms occurring with wandering supraventricular pacemaker with atrioventricular dissociation and with complete atrial capture In the latter instance the aberration was demonstrated during the recorded onset and offset of the nodal rhythm when on occasions curiously enough ventricular fusion beats were also noted As a general rule the aberration consisted primarily of slight changes in QRS configuration with little or no change in duration Ventricular fusion beats might possibly reflect simultaneous activation of the ventricles by impulses passing down the usual conducting pathways and by impulses arriving via the atrioventricular preferential pathways

The relative frequency with which nodal escape beats exhibit ventricular aberration and the confusion which may arise in differentiating these beats from those originating in the ventricles have been emphasized by Pick Moreover one is tempted to speculate whether this innate predilection of nodal beats to show aberration may not also apply to nodal tachyventricular aberration The answer to this question may be of therapeutic and prognostic significance

IDIOVENTRICULAR ESCAPE RHYTHM

When a secondary pacemaker located in the ventricles is permitted to escape as the result of either blocking of impulses from higher pacemaking centers or depression of the rhythmicity of these centers the ventricular rhythm produced is called an *idioventricular escape rhythm* (see Fig 311) This type of escape rhythm is observed chiefly in complete atrioventricular block when the site of the block is located relatively low in the atrioventricular junctional tissues and in instances of marked cardiac depression induced by such factors as quinidine intoxication hyperkalemia or terminal changes in the dying heart Inasmuch as idioventricular rhythms are described in some detail in a later discussion of the electrocardiographic findings in complete atrioventricular block only a brief preview of the electrocardiographic characteristics of idioventricular escape rhythms will be given at this point The salient features of this type of escape rhythm are as follows

1 When the ventricular pacemaker is situated above *the bifurcation of the common bundle* the ventricular deflections are generally indistinguishable from those produced by an atrioventricular nodal pacemaker

2 *Below the bifurcation of the common bundle* the generalization holds true that the more distal the *location of the pacemaker in the* intraventricular conduction system the more aberrant is the spread of excitation through the ventricles the greater is the distortion and widening of the idioventricular beats and the slower is the idioventricular pacemaker

3 As a general rule idioventricular rhythms are characterized by rates of 40 beats per minute or less and by QRS deflections which are bizarre in appearance and widened to 0 12 second or more

These interrelationships are responsible for *the fact* that coupled beats arising in the same ectopic *focus* (unifocal extrasystoles) tend to have the same appearance and essentially equal coupling intervals I [illegible] configuration and coupling

[illegible]

be kept in mind that variation in co[illegible] ectopic impulses may cause ectopic beats *from* a single focus to present the features of multifocal extrasystoles

Postectopic interval—The postectopic interval or pause *extends from onset of* the ectopic beat to onset *of the following beat of the prevailing rhythm* (see Fig 256) Usually the first postectopic beat is a sinus beat but occasionally the pause if prolonged is terminated by a nodal escape beat In such a case the postectopic interval corresponds to the time required for the atrioventricular node *to mature* and discharge an impulse

Compensatory (postectopic) pause—The postectopic interval is compensatory (see Fig 257) if the combined coupling and postectopic intervals (that is the interval between the sinus beat preceding and that following the ectopic beat) equal in length two cycles of the sinus rhythm (two R–R or P–P intervals)

Noncompensatory (postectopic) pause—The post ectopic pause is noncompensatory (see Fig 257) if the total length of the coupling and postectopic intervals *is shorter* than two sinus cycles but longer than a single R–R or P–P interval of the sinus rhythm

Interpo[illegible]—An interpolated extrasystole (F[illegible] single cyc[illegible] *not* prevent the following sinus beat from [illegible] *response* in contrast to the usual course of events Interpolated beats are almost always of ventricular or atrioventricular nodal origin interpolated atrial extrasystoles occurring only rarely

The many factors which in conjunction determine if an ectopic beat is to be interpolated or followed by a compensatory or noncompensatory pause can be reduced to a single variable—*namely the effect of the ectopic impulse if any on the sinus pacemaker or on* the following sinus impulse A *compensatory pause* results when the ectopic impulse fails to reach the sinus node in time to discharge it prematurely Instead it encounters the descending sinus impulse and each impulse obliterates the other As a result the ectopic beat does not disturb the rhythm of the sinus pacemaker and the sinus impulse does not produce a conducted beat The postectopic beat is therefore initiated by the next sinus impulse the second P wave following the extrasystole which appears at its expected

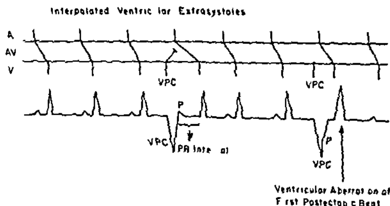


Fig 258 —Mechanism of interpolated extrasystoles An interpolated atrioventricular nodal or ventricular extrasys[illegible] in a single sinus cycle In other words each sinus P wave (P) elicits a ventricu[illegible]

atrioventricular conduction of the first postectopic sinus beat it is necessary to assume retrograde conduction of the ectopic impulse through a part of the atrioventricular junctional tissues which are therefore rendered partially refractory Thus in contrast to the usual course of events following a ventricular extrasystole the retrograde ectopic impulse of an interpolated ventricular extrasystole does not prevent atrioventricular conduction of the descending sinus impulse but simply delays it This is a manifestation of *concealed atrioventricular conduction* When the retrograde impulse of an interpolated ventricular extrasystole fails even to delay atrioventricular conduction of the first postectopic sinus beat as in the second example above the QRS deflection of the resulting conducted sinus beat generally shows aberration because the sinus impulse arrives in the ventricle while the intraventricular conducting pathways are still partially refractory following conduction of the preceding *ectopic beat*

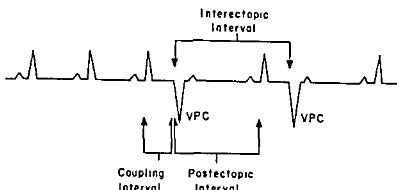


Fig 256 —Diagrammatic electrocardiogram depicting interectopic coupling and postectopic intervals The sinus beats are represented by upright P waves preceding upright R waves while ventricular premature beats (*VPC*) following the third and fourth sinus beats are downwardly directed The interval from onset of one ventricular premature beat to onset of the next ventricular premature beat is called the *interectopic interval* (If the extrasystolic beats were of atrial origin then the interectopic interval would extend from onset of the first premature ectopic P wave to onset of the next premature ectopic P wave) The *coupling interval* of the ventricular extrasystole is the interval between onset of the ectopic beat and onset of the QRS deflection immediately preceding it (In the case of an atrial extrasystole the coupling interval extends from onset of the ectopic P wave to onset of the sinus P wave immediately preceding the ectopic P wave) The *postectopic interval* extends from onset of the ventricular extrasystole to onset of the QRS deflection of the first postectopic sinus beat (The postectopic interval of an atrial extrasystole is the interval extending from onset of the premature atrial P wave to onset of the first postectopic sinus P wave)

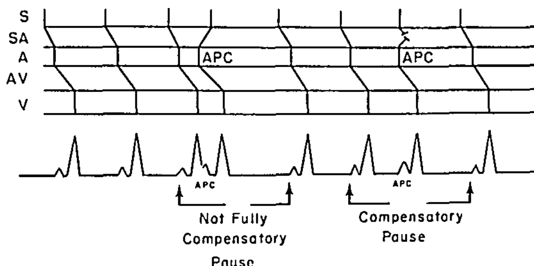
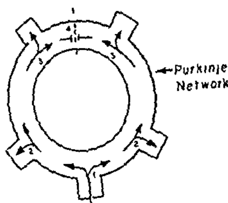
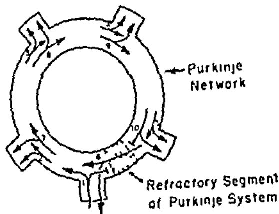


Fig 257 —Mechanisms of compensatory and noncompensatory pauses following extrasystole (Symbols S sinus node discharge *SA* sinoatrial conduction *A* atrial beat or P wave *AV* atrioventricular conduction *V* ventricular beat or QRS deflection and *APC* atrial premature contraction) In the schematic lead strip below the diagram the atrial premature beats (*APC*) are conducted into the ventricles to produce R waves having the same configuration as those of the sinus beats In the first example the atrial premature beat not only activates the ventricles but is able to spread in a retrograde direction and to discharge the sinus node prematurely The premature discharge of the sinus pacemaker causes a transient depression of its rhythmicity so that the sinus cycle immediately following the *APC* is slightly longer than the usual sinus cycle However the lengthening of the postectopic sinus cycle is usually less in

pulse is discharged at the expected
first postectopic sinus P wave is the
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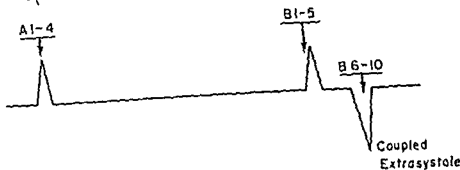


Fig 259 —Re-entry mechanism of premature ectopic beats In A a ring of conducting fibers ... Purkinje network are depicted The ...

... pulse re-enters the circular ring of conducting fibers and traverses it a second time to produce the coupled extrasystole (B 6-10)

of muscle distal to the site of blocking (this effect has been demonstrated in nerve preparations and has been termed *Wedensky facilitation* by neurophysiologists) Whether or not this is sufficient to produce suprathreshold excitability and discharge the ectopic focus is largely dependent on fluctuations in the subthreshold background

In the following discussion of ... opic ... was ... neo

... on any difference in the relative validity of the two concepts The fact is that neither the re-entry theory nor the theory of a single focus of subthreshold activity has been proved to the exclusion of the other insofar as the genesis of coupled ectopic beats is concerned (That re entry is the mechanism responsible for reciprocal beats proves nothing as to its role in the causation of coupled ectopic beats)

time Thus the interval between the sinus beats preceding and following the ectopic beat equals two cycles of the sinus rhythm Sometimes during a slow sinus rhythm ventricular or nodal extrasystoles occurring early in diastole may be *interpolated* between two consecutive sinus beats The reason for this is that the interval between onset of the ectopic beat and discharge of the next sinus impulse may be long enough to enable the conducting pathways and ventricular muscle to recover at least partially If such is the case the sinus impulse produces a ventricular beat Since the atrioventricular junctional tissues are still in the refractory phase following excitation by the ectopic beat they conduct the sinus impulse more slowly than normally Thus the P–R interval of the conducted sinus beat is prolonged and to this extent the R–R interval containing the interpolated beat exceeds in length the usual R–R interval These findings reflect incomplete atrioventricular interference between the ectopic and sinus impulses If atrioventricular interference is complete the sinus impulse is not conducted and the ventricular or nodal extrasystole is followed by a compensatory pause

A noncompensatory pause follows an extrasystole if the ectopic impulse is conducted in a retrograde direction to the sinoatrial node in time to discharge the immature sinus impulse before it can be fired off spontaneously Premature discharge of the sinus pacemaker which is the *sine qua non* for a noncompensatory pause has two major effects on the length of the postectopic interval (*a*) it shortens the sinus cycle preceding the ectopic beat and (*b*) premature discharge of the sinus node—or for that matter any impulse forming center—often depresses temporarily the rhythmicity of the pacemaker

More often than not sinus node depression produced in this manner lengthens the postectopic sinus cycle less than the preceding cycle is shortened The total duration of these two cycles is therefore less than two normal sinus cycles and so the postectopic interval is noncompensatory When marked prematurity of an ectopic beat or the occurrence of a succession of ectopic beats leads to a relatively more severe degree of sinus node depression the postectopic interval may be greatly prolonged and may be terminated by an atrioventricular nodal escape beat

COUPLED ECTOPIC BEATS

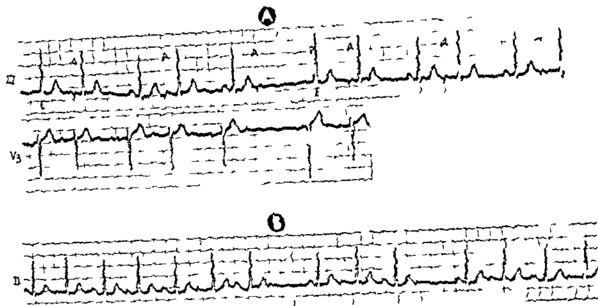
Mechanisms

The mechanism of coupled ectopic beats is still debated That a parasystolic center can on rare occasions produce both automatic and coupled beats is generally conceded However for all intents and purposes only two postulated mechanisms need be considered both of which assume focal origin of the ectopic impulse and its initiation by a preceding beat

Re entry—This mechanism (see Fig 259) assumes a focal depression of conductivity involving certain peripheral fibers of the muscle syncytium The initial sinus impulse finds these fibers still refractory from previous excitation The excitation impulse therefore enters other branches of the syncytial network which distribute it to muscle tissue elsewhere Meanwhile the depressed focus recovers its responsiveness Thus the sinus impulse wending its way through the muscle syncytium is led back into and passes along the previously blocked pathways By the time the previously depressed focus has undergone activation conducting fibers and muscle tissues excited initially may have emerged from the absolute refractory state with the result that the excitation impulse is picked up and transmitted once again through the myocardium Therefore the ectopic beat that is produced appears prematurely—that is it follows the preceding sinus beat by an interval which is shorter than a single cycle of the sinus rhythm

Single focus of subthreshold activity—According to Scherf a coupled beat is produced when a trigger sinus impulse (or another ectopic impulse) initiates discharge of an ectopic focus of subthreshold activity Presumably a localized metabolic disturbance causes a subthreshold increase in focal excitability If on this subthreshold background there is superimposed a further albeit temporary rise in focal excitability induced by the trigger impulse the level of excitability in the ectopic center becomes transiently suprathreshold This precipitates discharge of the ectopic impulse

The mechanism by which the trigger impulse increases excitability in the ectopic focus is not definitely known One possible explanation is that the initial beat causes circulatory changes locally which alter the chemical environment of the ectopic center On the other hand it may be that the initial beat increases subthreshold excitability by a mechanism related to the phenomenon of Wedensky facilitation Thus it is conceivable that a small focus of scar tissue or depressed muscle blocks completely or incompletely the spread of the trigger impulse into a small area of muscle tissue beyond Nevertheless the blocked impulse causes a momentary increase in the excitability



F g 261 —Coupled atrial extrasystoles wi

having the same configuration as the deflections initi ated by sinus beats The exceptions to this rule are discussed below

Variations in Atrioventricular and Intraventricular Conduction

An ectopic atrial beat with a short coupling interval sometimes may be of such premature onset that it appears in early ventricular diastole At this time the atrioventricular and or intraventricular conducting pathways may still be completely refractory from the preceding sinus beat When this occurs the premature atrial beat fails to produce a ventricular deflection The atrial extrasystole often referred to as a *blocked extrasystole* is better described as "nonconducted if it occurs within the Q–T interval of the preceding beat (that is within the normal refractory period of the atrioventricular junctional tissues and/or intraventricular conducting pathways) since the failure of the ectopic atrial impulse to be transmitted into the ventricles can be ascribed in this instance solely to atrioventricular interference without the need of implicating pathologic blocking If the atrial ectopic beat appears outside the Q–T interval of the preceding beat and yet fails to be conducted then the atrial premature contraction can justifiably be said to be *blocked* (Fig 261) On the other hand the atrioventricular and/or intraventricular pathways may have partially recovered before arrival of the ectopic atrial impulse Although the impulse is transmitted the refractoriness of the conducting tissues leads to prolonged atrioventricular conduction and/or aberrant intraventricular conduction Generally the longer the P–R interval of the atrial extrasystole the less likely it is that the QRS complex will show ventricular aberration or the less marked the aberration The reason for this is that the prolonged atrioventricular conduction lengthens the time available to the intraventricular conducting fibers for recovery When present ventricular aberration may consist of either slight changes in duration and/or amplitude of the Q R and S waves or the appearance or disappearance of one of these waves At the other extreme the changes may sometimes be so striking that the QRS pattern resembles that of right bundle branch block (in 85% of the affected beats) (Fig 262) or left bundle branch block A ventricular deflection with aberration of this degree may be readily confused with a ventricular extrasystole if the preceding P wave of

Coupled Ectopic Atrial Beats

Electrocardiographic Appearance

The excitation impulse producing an atrial extrasystole or coupled beat originates in an ectopic focus in the atria (Fig 260) and in consequence its spread through the atria differs from that of the impulses arising in the sinoatrial node The abnormal spread of excitation and its early onset produces a premature P wave which differs from the sinus P wave in appearance Moreover ectopic atrial impulses arising from several foci spread through the atria differently and vary in P wave configuration one from another The nearer their site of origin to the sinoatrial node the more they resemble sinus P waves Conversely the nearer to the atrioventricular node the ectopic atrial beat arises the greater its similarity to the retrograde P wave of a nodal beat If leads II III and/or aVF record a premature inverted P wave which is followed by a ventricular deflection after a P–R interval exceeding 0 12 second the likelihood is that the P wave represents an ectopic atrial or coronary sinus beat although a nodal premature systole with prolonged antegrade atrioventricular conduction cannot be excluded Between these two extremes there can be seen atrial extrasystoles with a low isoelectric or diphasic P wave configuration As a general rule ectopic atrial beats originating in the same focus show essentially the same P wave configuration and have approximately equal coupling intervals (measured from sinus P wave to ectopic P wave) However in a given instance the correlation between the appearance of an ectopic atrial beat and its site of origin is uncertain because variations in intra atrial conduction may cause unifocal atrial extrasystoles to simulate ectopic beats of multifocal origin Ordinarily an atrial extrasystole produces a ventricular deflection

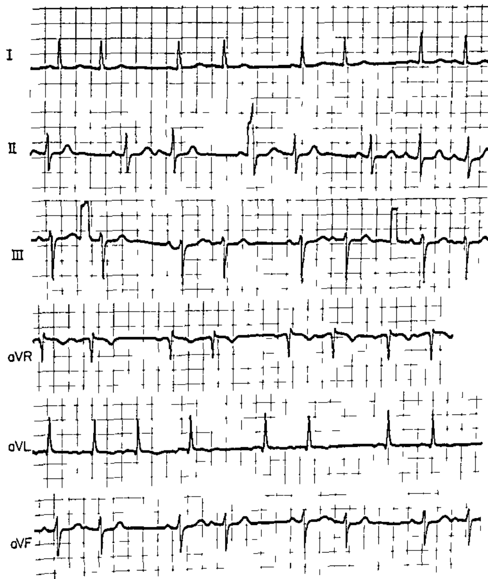


Fig 260 – Bigeminal rhythm due to a coupled atrial extrasystole following every sinus beat The close similarity of the ectopic and sinus P waves suggests that the atrial extrasystoles originate in a focus near the sinoatrial node The ventricular complexes following the premature P waves do not show aberration

the premature atrial beat is overlooked Since the ectopic P wave is often buried in the previous T wave the latter must be examined carefully for slurring notching or any other irregularity of its contour which might indicate a superimposed atrial beat (Fig

tion can be attributed to the prematurity of the ec-op atrial beat. If these abnormalities accompany a premature atrial beat appearing late in electrical diastole—particularly if onset of the ectopic beat occurs well after the preceding T wave—a latent atrioventricular or intraventricular conduction defect may be impli , , , prolonged atrioventricular conduc-

,

by a lengthened refractory period and so a coupled atrial beat may find the atrioventricular node and/or intraventricular conduction system only partially recovered. Thus the QRS complex follows the ectopic P wave by a prolonged P–R interval or displays ventricular aberration However other atrial extrasystoles

sponsible for conduction disturbances involving the first of a run of ectopic atrial beats The remaining beats are conducted normally because the premature onset of the initial extrasystole decreases the cycle length and shortens the refractory period for the extrasystoles following Similarly only the second of two coupled atrial beats may evidence disturbed atrioventricular and or intraventricular conduction the reason being that its initiating sinus beat follows the post ectopic pause of the preceding extrasystole

The Postectopic Interval

Ectopic atrial impulses are conducted so rapidly to the sinoatrial node that ordinarily they discharge it prematurely Premature discharge of the sinus node not only shortens the cycle preceding the extrasystole but also by depressing the sinus pacemaker slows formation of the next sinus impulse Consequently the first sinus P wave after the ectopic beat is delayed somewhat in onset and the postectopic interval exceeds the length of a single sinus cycle Nonetheless the total duration of the pre- and postectopic P–P intervals is shorter than two sinus cycles The post ectopic pause is therefore noncompensatory

Sometimes the impulse producing the first post ectopic atrial beat originates elsewhere than in the sinus node In fact the P wave configuration of such a beat often resembles that of the previous atrial extrasystole A postectopic beat of this type has been ascribed by some to a shift of the cardiac pacemaker to another site Other investigators propose that the initial atrial extrasystole precipitates formation of another ectopic beat in the same or a different atrial focus which then discharges spontaneou

minat

sinus -

vals terminated by sinus beats If sinus node depression is quite marked nodal escape may occur but in contrast to the postectopic atrial extrasystolic P wave described above a P wave preceding the nodal escape beat if present is almost invariably a non

discharge spontaneously before the ectopic impulse can penetrate the sinoatrial junction Such a condition may have any one of the following causes (a) the atrial impulse may be discharged too late in diastole to reach the sinus node before it fires off spontaneously (b) retrograde conduction of the ectopic impulse from its site of origin to the sinoatrial junction may proceed too slowly in comparison with the rate of forward conduction of the sinus impulse from the sinoatrial node or (c) the rate of impulse formation and discharge by the sinus pacemaker may be too rapid Any of these factors may lead to sinus and ectopic atrial impulses meeting and interfering with each other in or near the sinoatrial junction as evidenced by the fact that the ectopic impulse activates the atria without interrupting the sinus pacemaker Thus the postectopic sinus P wave appears at the expected time and follows a compensatory pause Sometimes the sinus and ectopic impulses do not meet until each has excited a portion of the atria In this event interference occurs not in the sinoatrial junction but in the atrial muscle Because of the occurrence of atrial interference a fusion P wave is observed rather than the P wave of the ectopic atrial beat The configuration of the fusion beat is intermediate between that of sinus P waves and ectopic P waves elsewhere in the same lead and its onset approximates the expected time of onset of the next sinus P wave

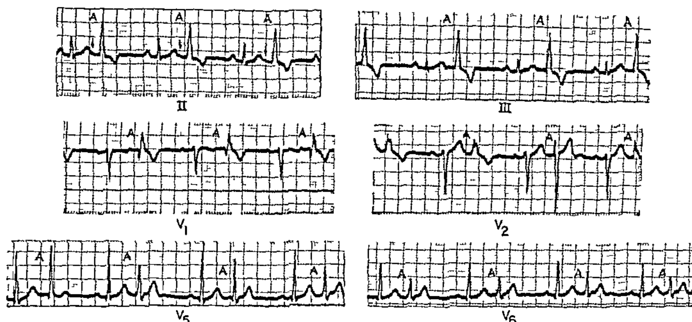
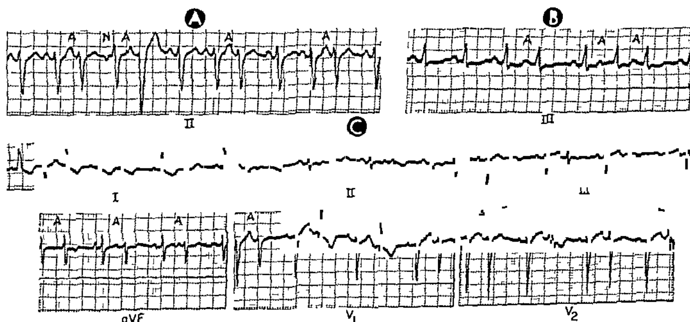


Fig 262 —Coupled ectopic atrial beats giving rise to a bigeminal rhythm In these lead strips the premature ectopic atrial beats are indicated by *A* The QRS deflections produced by the premature atrial beats show ventricular aberration in the form of a right bundle branch block type of configuration best demonstrated in lead V_1

Fig 263 —Coupled ectopic atrial beats (*A*) with and without ventricular aberration of the following QRS complexes The inverted P wave labeled V in lead *II* of **A** is an atrioventricular nodal extrasystole



the premature atrial beat is overlooked Since the ectopic P wave is often buried in the previous T wave the latter must be examined carefully for slurring notching or any other irregularity of its contour which might indicate a superimposed atrial beat (Fig

tion can be attributed to the prematurity of the ectopic atrial beat. If these abnormalities accompany a premature atrial beat appearing late in electrical diastole—particularly if onset of the ectopic beat occurs well after the preceding T wave—a latent atrioventricular or intraventricular conduction defect may be impli [illegible] longed atrioventricular conduc

ectopic impulse and the presence of a latent [illegible] tion defect This mechanism is based on the fact that the recovery curve of heart muscle for a given cycle is [illegible] the preceding cycle A

atrial beat may find the atrioventricular node [illegible] intraventricular conduction system only partially re [illegible]

with the same coupling interval are conducted normally because their initiating sinus beats are preceded by shorter R–R cycles This mechanism is often responsible for conduction disturbances involving the first of a run of ectopic atrial beat The remaining beats are conducted normally because the premature onset of the initial extrasystole decreases the cycle length and shortens the refractory period for the extrasystoles following Similarly only the second of two coupled atrial beats may evidence disturbed atrioventricular and or intraventricular conduction the reason being that its initiating sinus beat follows the postectopic pause of the preceding extrasystole

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Sometimes the impulse producing the first postectopic atrial beat originates elsewhere than in the sinus node In fact the P wave configuration of such a beat often resembles that of the previous atrial extrasystole A postectopic beat of this type has been ascribed by some to a *shift of the cardiac pacemaker to another site Other investigators propose that the initial atrial extrasystole precipitates* formation of another ectopic beat in the same or a different atrial focus which then discharges spontaneously In [illegible] minated by P w[illegible] sinus P waves [illegible] vals terminated by sinus beats If sinus node depression is quite marked nodal escape may occur but in contrast to the postectopic atrial extrasystolic P wave described above a P wave preceding the nodal escape beat if present is almost invariably a nonconducted sinus P wave

Occasionally an atrial extrasystole may be followed by a compensatory pause if the sinus node is able to discharge spontaneously before the ectopic impulse can penetrate the sinoatrial junction Such a condition may have any one of the following causes (*a*) the atrial impulse may be discharged too late in diastole to reach the sinus node before it fires off spontaneously (*b*) retrograde conduction of the ectopic impulse *from its site of origin* to the sinoatrial junction *may proceed too slowly* in comparison with the rate *of forward* conduction of the sinus impulse from the *sinoatrial node* or (*c*) the rate of impulse formation and discharge by the sinus pacemaker may be too rapid Any of these factors may lead to sinus and ectopic atrial impulses meeting and interfering with each other in or near the sinoatrial junction as evidenced by the fact that the ectopic impulse activates the atria without interrupting the sinus pacemaker Thus the postectopic sinus P wave appears at the expected time and follows a compensatory pause Sometimes the sinus and ectopic impulses do not meet until each has excited a portion of the atria In this event interference occurs *not in the sinoatrial* junction but in the atrial muscle *Because of the* occurrence of atrial interference a fusion P wave is observed rather than the P wave of the ectopic atrial beat The configuration of the fusion beat is intermediate between that of sinus P waves and ectopic P waves elsewhere in the same lead and its onset approximates the expected time of onset of the next sinus P wave

Coupled Ectopic Atrioventricular Nodal Beats

Electrocardiographic Appearance

An ectopic impulse arising in the atrioventricular node is conducted through the junctional tissues in two directions simultaneously—namely, in a retrograde direction toward the atria and in an antegrade or forward direction toward the ventricles. A nodal extrasystole therefore consists of a premature ventricular deflection which resembles or differs only slightly in appearance from conducted sinus beats and which precedes, follows, or coincides with a retrograde P wave (inverted in leads II, III, and aVF and upright in lead aVR). The relative positions of the retrograde P wave and QRS complex of a nodal beat are determined by the time relationship between onset of ventricular excitation and onset of atrial excitation. Whether the ventricles receive the excitation impulse before the atria or vice versa is a function of two variables—namely, (*a*) the site of impulse origin in the atrioventricular node and (*b*) the comparative rates of antegrade and retrograde atrioventricular conduction. The general features of nodal beats were described fully in the section on atrioventricular nodal rhythms (pp. 363–367) and will only be summarized at this point.

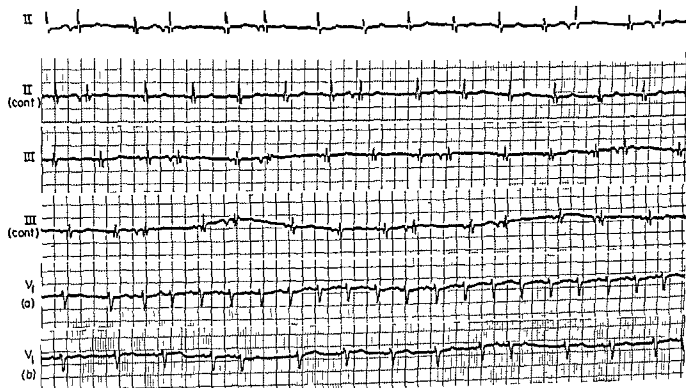
Upper atrioventricular nodal beats.—The retrograde P wave, which is inverted in leads II, III, and aVF and upright in lead aVR, precedes the ventricular deflection by a P–R interval of 0.12 second or less. (If the P–R interval is longer than 0.12 second, there may be delayed forward conduction of the upper nodal impulse, or the ectopic beat may originate near the coronary sinus area in the atria.) (Fig. 264.)

Middle atrioventricular nodal beats.—The retrograde P wave is buried in and obscured by the ventricular complex of the nodal beat.

Lower atrioventricular nodal beats.—The retrograde P wave follows the QRS complex by an R–P interval of 0.10–0.20 second.

Variations in Atrioventricular and Intraventricular Conduction

Coupled atrioventricular nodal beats may show evidence of disturbed atrioventricular and/or intraventricular conduction for reasons previously cited—namely, (*a*) markedly premature onset of the nodal



there is a short bout of paroxysmal atrioventricular nodal tachycardia

beat (*b*) latent atrioventricular and/or intraventricular conduction defects unmasked by the shortened recovery period following the initial sinus beat and (*c*) a prolonged refractory period *after the initial* sinus beat when this beat is preceded by a *long R–R* interval.

Variations in atrioventricular conduction of nodal impulses have been discussed previously and will not be considered in detail in this section *In brief recapitulation*, disturbed retrograde and/or antegrade atrioventricular conduction of a nodal impulse may be accompanied by any of the following findings

1 If retrograde atrioventricular conduction is *completely blocked* the nodal impulse produces only a premature ventricular deflection which is not accompanied by a retrograde P wave while a premature retrograde P wave without an associated QRS complex is indicative of blocked antegrade conduction.

2. Delayed retrograde or antegrade atrioventricular conduction of a nodal impulse may be suspected

differ from the P–QRS relationship present in other nodal extrasystoles this may be a manifestation of disturbed atrioventricular conduction or it may signify that the nodal extrasystole in question arises from a different focus In general prolonged retrograde *conduction shifts the retrograde P* wave behind or farther behind, the ventricular deflection so that the R–P interval lengthens Delayed antegrade conduction causes the QRS complex to lag farther behind the retrograde P wave the P–R interval increasing

The previous discussion of ventricular aberration of ectopic atrial beats pertains equally well to aberrant intraventricular conduction of nodal extrasystoles and need not be repeated

The Postectopic Interval

If the retrograde atrioventricular nodal impulse discharges the sinus node prematurely the postectopic interval of the nodal extrasystole is longer than a *single* sinus cycle but is noncompensatory as *previously explained* with reference to atrial extrasystoles A compensatory postectopic pause is more frequently

delayed or blocked retrograde conduction A retrograde nodal impulse may fail to discharge the sinus node for either of the following reasons (*1*) there *may* be a unidirectional atrioventricular block which *involves retrograde* conduction but does not disturb *antegrade* conduction and (2) the sinus impulse and the retrograde nodal impulse may interfere with each other at any of the following sites

1 *Atrioventricular interference*—If interference occurs in the atrioventricular junctional tissues *the* ventricular complex alone is produced by the nodal impulse while the normal sinus P wave appears at its expected time but is not conducted

2. *Atrial interference*—If the sinus and retrograde impulses do not meet until each has activated a portion of the atrial muscle the QRS complex of the nodal beat is preceded or followed by a fusion P wave

3 *Sinoatrial interference*—If the retrograde nodal impulse is able to complete activation of *the atria before* meeting the descending sinus impulse very near or in the sinoatrial junctional tissues the only evidence that interference has occurred will be the compensatory postectopic pause In short the nodal beat controls both the atria and ventricles but does not interrupt the sinus pacemaker Thus the postectopic sinus P wave appears at the time anticipated

Coupled Ectopic Ventricular Beats

Electrocardiographic Appearance

Ventricular extrasystoles appear in the electrocardiogram as premature QRS deflections which are widened to 0 12 second or more are slurred and of large *size* and are not preceded by either ectopic or *conducted* sinus P waves These alterations in QRS configuration and duration reflect asynchronous onset of excitation in the two ventricles and aberrant spread of the excitation impulse subsequently through the myocardium The more pronounced the disturbance of ventricular excitation the more abnormal is the appearance of the ventricular deflection The site of origin of the ectopic ventricular impulse relative to the location of the normal intraventricular conducting pathways affects the manner in which excitation spreads through the ventricles and therefore *influences* the appearance of the ventricular extrasystole The following general relationships can be accepted with some reservations

1 Ectopic *impulses* arising in the bundle of His above its bifurcation utilize the normal intraventricular conduction system Consequently they produce ventricular complexes which have the same appearance *as conducted sinus* beats

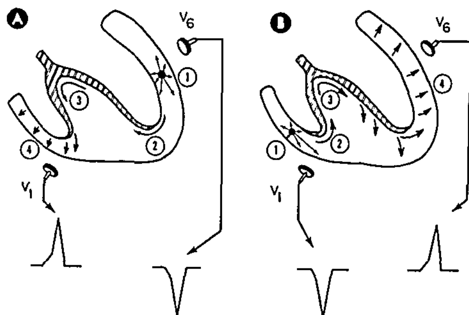


Fig 265 —Localization of the site of origin of ventricular extrasystoles In A the site of ectopic impulse origin is assumed to be in the left ventricular wall at point *1* The ectopic impulse spreads in an aberrant manner through the left ventricular myocardium and eventually *enters the left bundle branch at 2 It is transmitted in a retrograde direction* up the left bundle branch and then turns down the right bundle branch (*3*) and is distributed to the right ventricle in a normal manner (*4*) Thus the over all spread of ectopic ventricular excitation occurs in a left to right direction so that

Thus when a ventricular extrasystole originates in the right ventricle the over all direction in which it spreads is from right to left causing lead V_1 to record a downwardly directed ventricular beat and lead V_6 to register an upright ventricular extrasystole It must be stressed that this explanation is greatly oversimplified and does not permit precise localization of the site of origin of a ventricular extrasystole

2 An *excitation impulse originating more distally* in the conducting system first spreads through adjacent myocardium and the Purkinje network to activate initially the ventricle in which it arises (Fig 265) In so doing the ectopic impulse enters and traverses in a retrograde direction the ipsilateral bundle branch On reaching the bifurcation the excitation impulse then turns back into the opposite bundle branch and is distributed to and activates the other ventricle via the normal conducting pathways Thus the more distal in the conducting system the ectopic impulse originates the more aberrant is the spread of excitation through the ipsilateral ventricle and the more delayed the onset of excitation in the contralateral ventricle For example ectopic impulses arising in the free wall of the left ventricle produce ventricular deflections of longer duration and more abnormal configuration than impulses originating in the interventricular septum However in addition to the site of impulse origin other factors—such as myocardial *disease* variations in intraventricular conduction and heart position—*may influence the appearance of ventricular extrasystoles according to some authorities* For example ectopic ventricular beats with a QRS duration exceeding 0 16 second are thought to signify underlying cardiac disease On the other hand ectopic beats with QRS intervals less than 0 16 second neither support nor refute the possibility of cardiac pathology

3 The excitation process in bundle branch block and that of ventricular extrasystoles are similar in at least one respect each produces resultant electrical forces which on the average are directed toward the ventricle last activated Thus if the ectopic ventricular impulse is distributed first to the left ventricle the QRS vectors are rotated toward the right ventricle and produce large positive deflections in right precordial leads and resultantly negative complexes in *left precordial leads If the ventricular extrasystole* arises in the right ventricle the QRS vectors of the ectopic beat are directed toward the left ventricle and project positivity on left precordial leads and negativity on right precordial leads Inasmuch as the sequence of ventricular repolarization is altered be

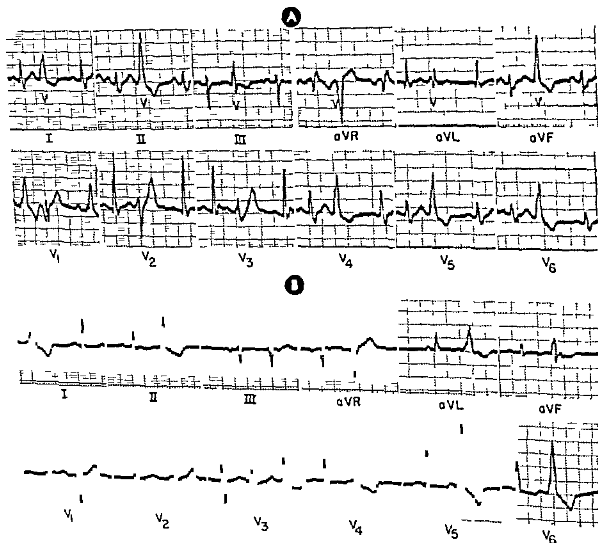
cause of changes in depolarization the ST-T vectors tend to be directed away from the QRS vectors Thus ectopic ventricular beats which are resultantly positive are usually followed by inverted T waves with S-T segment depression and downwardly directed ventricular deflections by upright *T waves with S-T* segment elevation These findings represent so-called *secondary T wave changes*

4 In a given lead ventricular extrasystoles with the same site of origin generally have essentially equal

ls and usually exhibit much the same

tervals Multiform extrasystoles may reflect variations in intraventricular conduction of impulses arising in a single focus Ectopic ventricular beats induced by digitalis are frequently of the multiform type

Fig 266 —I
the twelve leads
the conducted beats are diagnostic of rig
coupling intervals The nonconducted sinus



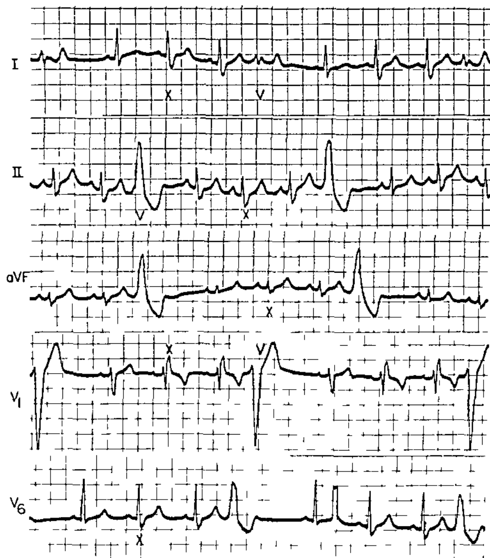


Fig 267 —Quadrigeminy due to ventricular extrasystoles with conducted sinus beats showing intermittent right bundle branch block Every third conducted sinus beat is followed by a ventricular extrasystole (*V*) probably originating in the right ventricle The pauses following the extrasystoles are fully compensatory and the first postectopic sinus beats are usually normal in appearance and duration However the second (X) and third postectopic sinus beats in each lead are diagnostic of right bundle branch block Evidently the compensatory pause following each ventricular extrasystole permits the intraventricular conducting pathways to recover completely so that the first postectopic sinus beat is normal in appearance But the following two sinus beats are preceded by shorter cycles and therefore arrive in the ventricles while the right bundle branch block is refractory following conduction of the previous beat the result being right bundle branch block

Retrograde Atrioventricular Conduction and the Postectopic Interval

In the past retrograde conduction of impulses originating in a ventricular focus was thought to be a rare occurrence More recently the fact has become apparent that retrograde activation of the atria by ectopic ventricular impulses occurs far more frequently than commonly realized The reason this event is not more frequently recognized is that retrograde P waves are often difficult to detect because they are superimposed on some portion of the ectopic ventricular beat *On the other hand a sinus* P wave distorted by part of the ectopic ventricular beat can readily be mistaken for a retrograde P wave As an aid to the correct identification of retrograde P waves accompanying ventricular extrasystoles the following criteria have been proposed

1 The P wave must be of premature onset with reference to the sinus P–P *interval*
2 The interval between retrograde P wave and the next sinus P wave must be longer than a single sinus P–P interval
3 There must be unequivocal inversion of the P waves *in lead II preferably* and/or in leads III and aVF
4 The retrograde P wave must follow the QRS complex of the ectopic beat by an interval which equals or exceeds the P–R interval of conducted sinus beats elsewhere in the electrocardiogram
5 If present in a given lead retrograde P waves tend to follow the ventricular extrasystoles by R–P intervals of nearly equal length

That retrograde activation of the atria by an ectopic ventricular impulse does not occur more often than is noted is not surprising in view of the long pathway the impulse must traverse to reach the atria Thus an ectopic ventricular impulse unlike a nodal impulse must pass through the entire length of the

atrioventricular node the most time-consuming part of the retrograde pathway. *Consequently it is not* often that the ventricular impulse gets the opportunity to activate the atria before a sinus *impulse can* interfere and even less often is the ventricular impulse able to discharge the sinus node prematurely. On the contrary the next sinus impulse almost *always* interferes with retrograde spread of the ectopic im*pulse at one of the following levels*

Sinoatrial interference.—Interference between sinus and ectopic *impulses in the sinoatrial* junctional tissues can be inferred if the ventricular extrasystole is followed by a retrograde P wave and a compensa*tory pause*

Atrioventricular interference.—In most instances interference occurs between the two impulses in or near the atrioventricular junction (Figs 268 and 269) The sinus impulse activates the atria and the ectopic impulse activates the ventricles but neither penetrates the atrioventricular node completely. The *sinus P wave ordinarily is buried in the ventricular* extrasystole although sometimes it can be identified in the initial portion of the QRS deflection or superimposed on the S-T segment or T wave

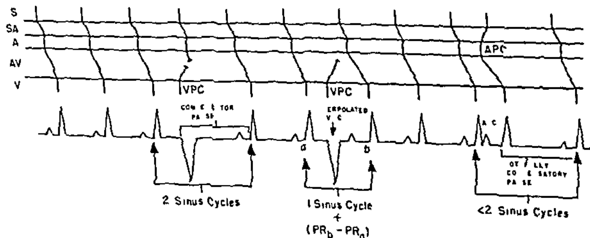
Atrial or ventricular interference.—Interference in the atria between a sinus impulse and a retrograde *ventricular* impulse is indicated in the electrocardiogram by the presence of a fusion P wave following a *ventricular* extrasystole If either the coupling intervals of ventricular extrasystoles occurring late in di*astole* tend to vary or if there is sinus arrhythmia an *occasional sinus impulse may arrive in the ventricles* at the approximate time an ectopic ventricular impulse is *discharged* Each impulse activates part of the myocardium

refractory musc

impulses meet

ess of ventricular interference *The sinus initiated* excitation process *which spreads through the myo*cardium in a normal manner and the ectopic excitation process with its erratic aberrant spread both contribute to the configuration of the ventricular deflection, thereby producing a ventricular fusion beat

Occasionally in slow sinus rhythms the ectopic ventricular beat appears so early in diastole that the conducting pathways and ventricular muscle are able to recover sufficiently to respond to the next sinus impulse The postectopic sinus impulse which as explained above usually fails to be conducted in this instance initiates ventricular excitation The ventricu



ventricular interference with the *descending* sinus impulse the basic sinus rhythm is not disturbed Consequently the R R interval containing the ventricular premature beat *equals twice the basic R–R* cycle length. The next ventricular extrasystole is interpolated Thus the retrograde ectopic *ventricular impulse* interferes *with the descending* sinus impulse in the atrioventricular junction *only to the extent* that it causes prolongation of atrioventricular conduction of the

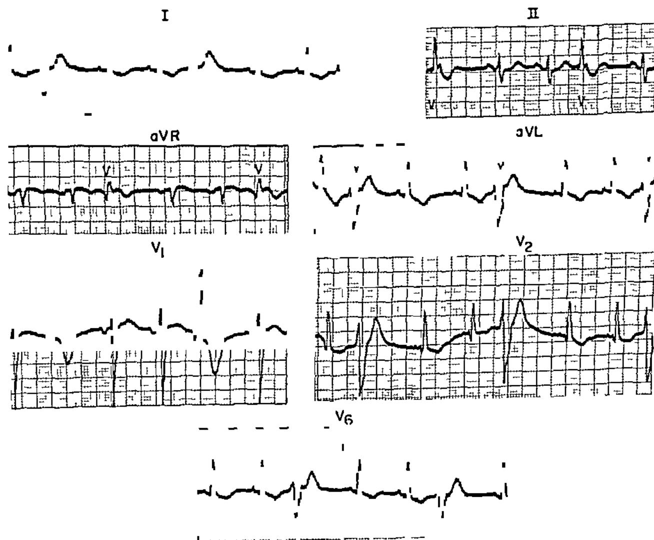
terval containing the atrial extrasystole are less than two sinus cycles in length.

lar extrasystole is therefore included in a single R–R interval—that is to say it is interpolated (Figs 270 and 271) Because of the very short postectopic interval of the interpolated beat the intraventricular conducting pathways and the myocardium may still be partially refractory when the sinus impulse arrives As a result the postectopic sinus beat may sometimes show ventricular aberration Another and more common finding is prolongation of the P–R interval of the postectopic sinus beat The partial refractoriness of the atrioventricular junctional tissues following an interpolated ventricular extrasystole as evidenced by the slower rate of atrioventricular conduction indicates that the retrograde ectopic impulse must have penetrated the atrioventricular node almost completely without emerging from it In other words *concealed atrioventricular conduction* of the ectopic beat has occurred Since the sinus impulse discharged after the interpolated beat is conducted more slowly than the sinus impulse preceding the ectopic beat the interval between them is generally longer than a single sinus cycle

Postectopic T Wave Changes

Occasionally the T wave of a sinus beat terminating a long postectopic pause shows changes in configuration voltage and/or polarity Less frequently T wave changes are observed in as many as three or four of the conducted sinus beats following an extrasystole Inverted T waves can become upright in the postectopic beat upright T waves usually become low or inverted or sometimes simply gain in amplitude The mechanism of postectopic T wave alterations is not definitely known but seems to be related in some way to the long pause preceding the post

Fig 269 —Trigeminal rhythm due to single ventricular extrasystoles (V) of left ventricular origin following every two conducted sinus beats



V
V P
V P
A II
B II
V₁

V P
V P
V P
C V₁
V₃
V₅

V
N P
D II
E II

V P
V P
Compensatory Pause
F V₁

A F cre recorded from different patients but all show

in the above lead strips originate in the ventricles in leads
sinus beat

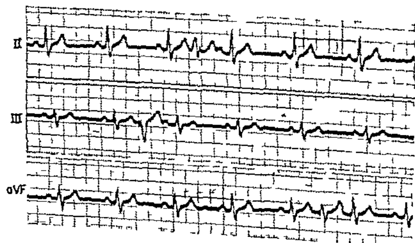


Fig 271 —Interpolated ventricular extrasystoles with prolonged atrioventricular conduction of the first postectopic sinus beat

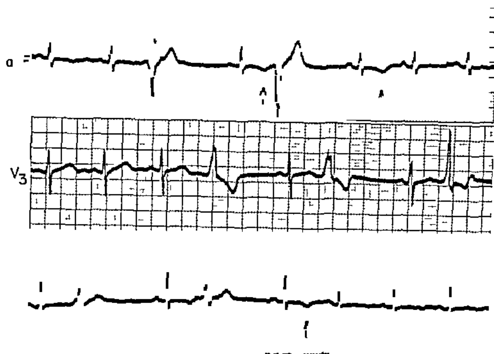


Fig 272 —Postextrasystolic T wave changes In lead aVF the T waves following the conducted sinus beats are generally diphasic or essentially isoelectric However the T waves of the first postectopic sinus beats following each of the ventricular extrasystoles (V) are inverted This finding represents a postextrasystolic T wave change In lead V the T waves following the sinus beats in the right half of the lead strip are lower than the T waves following the consecutive sinus beats on the left These changes as well as the T wave inversion indicated by the arrow in lead V also represent postextrasystolic T wave changes

ectopic sinus beat This *association is demonstrated* by the fact that interpolated extrasystoles (without a *postectopic pause) never produce T wave changes* while on the other hand altered T waves may occa*sionally follow long pauses in atrial fibrillation or* atrial premature beats Various clinical studies of the *phenomenon* of postectopic T wave change are in general agreement as to its high incidence in patients *with* cardiac disease in contrast to its relative rarity in normal subjects (Figs 272–274)

Clinical Significance of Coupled Ectopic Beats

The sporadic occurrence of *coupled ectopic beats* in normal subjects is relatively common particularly *if predisposing influences are present* (excess coffee tea and tobacco emotional disturbances etc) Con*sequently the greater frequency with which* extrasystoles occur in patients with diseased hearts is of limited value *in establishing the significance* of ectopic beats in a given person The presence of extrasystoles *is not in itself* indicative of *underlying myocardial* damage but certain features of the premature beats have an approximate correlation with the cardiac status of the patient For example

1 *Extrasystoles which occur frequently and have* a multifocal origin (based on their differing coupling *intervals and appearance) are usually observed in* patients with cardiac disease sometimes as the result *of digitalis toxicity*

2 If ectopic beats become more frequent during and immediately after exercise coronary insufficiency and/or myocardial damage are likely to be present *Tachycardia* exercise induced or *otherwise tends to* abolish ectopic beats in normal subjects but its failure to do so cannot be construed as evidence of cardiac abnormality

3 Some authorities attach much the same significance as the above to the occurrence of extrasystoles during rapid sinus rhythm or to the presence of both atrial and ventricular extrasystoles in the same electrocardiogram

4 A bigeminal rhythm consisting of an ectopic beat usually a *ventricular extrasystole coupled to* every sinus beat is commonly observed in digitalis intoxication *Myocardial disease is probably an additional* causative factor in most instances since it has been proved *almost impossible to produce a bigeminal* rhythm which is due to ventricular extrasystoles in a normal subject even *with large amounts of digitalis*

5 A finding considered almost pathognomonic of

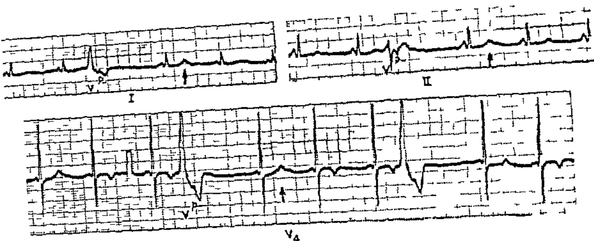


Fig 273 —Postextrasystolic T wave changes In leads I and II the upright T wave of the first postectopic sinus beats (indicated ... T ... e of the first postectopic beat (indicated by an arrow) is ... of the ve... the ectopic ventricular impulses into the atria

Fig 274 —Postextrasystolic T wave changes in t... In A the postextrasystolic T wave changes in leads II ... beats than is present in sinus beats else ... inversion

A

II

V_3

V_5

V_6

B

V_5

severe digitalis intoxication is the presence of bidirectional ventricular extrasystoles. These appear as paired oppositely directed ectopic ventricular beats, the QRS complex of one being resultantly positive and the other resultantly negative.

6. Frequent multifocal ectopic beats arising in the atria may precede onset of supraventricular tachycardia, atrial flutter or fibrillation. Similarly, extrasystoles of ventricular origin may herald onset of paroxysmal ventricular tachycardia, flutter or fibrillation, particularly if the ectopic beats are superimposed on the preceding T waves. More will be said of this in a later discussion of ectopic ventricular tachycardia.

7. In other clinical syndromes extrasystoles are sometimes an early sign of secondary cardiac involvement. Usually, however, such a wide variety of extracardiac factors are potentially involved, some capable of producing directly or indirectly ectopic beats, that the significance of the extrasystoles in a given case cannot be clearly defined in the absence of other evidence as to the cardiac status of the patient.

AUTOMATIC ECTOPIC BEATS (PARASYSTOLE)

Parasystole consists of the simultaneous activity of two independent impulse forming centers, one of which is protected from the other and each competing with the other to activate the atria and/or ventricles. The ectopic parasystolic pacemaker is usually situated in the ventricles (rarely in the atria or atrioventricular node), while the competing rhythm generally emanates from the sinoatrial node. Atrial and atrioventricular nodal types of parasystole will not be described (although examples are presented in subsequent figures), since the ventricular form is far more common and more clearly illustrates the parasystolic mechanism. Nevertheless, the general principles are the same irrespective of the location of the parasystolic pacemaker.

Parasystolic ventricular beats differ from coupled

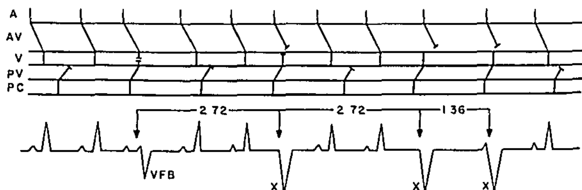


Fig. 275.—Mechanism of ventricular parasystole. (Symbols: *A*, atrial beats or P waves; *AV*, atrioventricular junctional tissues; *PC*, parasystolic center; *PV*, junctional tissues between the parasystolic center and the ventricular myocardium; *V*, ventricular beats; *VFB*, ventricular fusion beat; and X, ectopic ventricular beats produced by the parasys

tricular myocardium simultaneously with the sinus impulse so that the resulting QRS complex is a fusion of the normal

the pacemaker and surrounding ventricular myocardium and to enter the ventricular myocardium before the arrival of the conducted sinus impulse

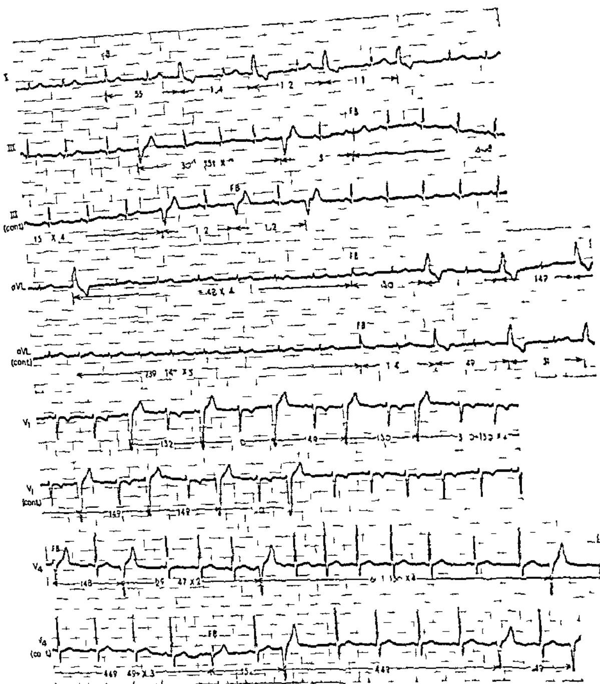


Fig 276.—Ventricular parasystole (Symbol *FB* ventricular fusion beat) In these lead strips the ectopic ventricular beats show varying coupling intervals but the interectopic intervals are equal to or are whole number multiples of the basic parasystolic cycle length of approximately 1.52 second In the continuous leads strips of lead aVL, the parasystolic cycle length is shortened to 1 47–1 49 second Ventricular fusion beats *are present in* lead I and in both strips of lead III

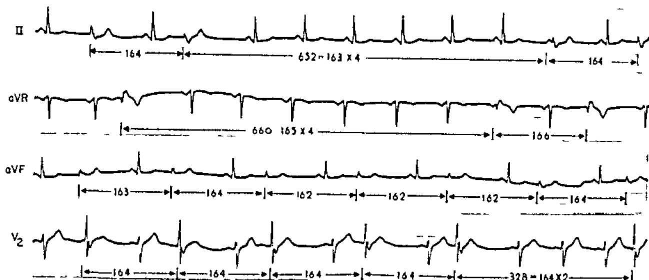


Fig 277 —Ventricul ir p irasystole Note the w
yet the relatively constant interectopic interv ils ı
ıll longer interectopic intervals are whole numbe
in lead aVR may possibly be ı ventricular fusion be ıt

Fig 278 —Atrioventricul ir nodal parasystole In atrioventricular nodal parasystole ventricular fusion beats are obviously not seen However the varying coupling interv ıls and the relatively constant lengths of the measured or cal culated parasystolic cycles are compatible with a parasystolic mech ınism of the ectopic nodal ventricular beats The lead strip in **B** was recorded several days after the lead strips in **(A)** but the parasystolic cycle length is relatively un changed

A

I 218 = 109 X 2 784

I (cont) 112 X 7 555 = 111 X 5 222 = 111 X 2

II

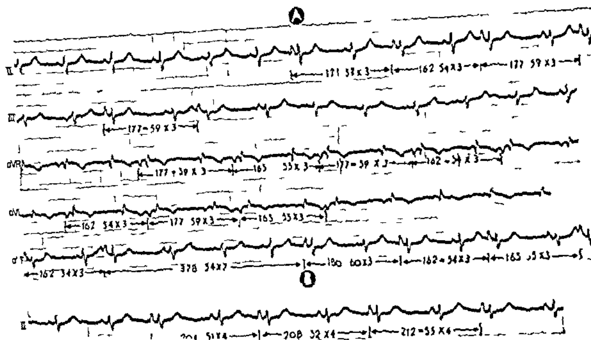
V5

V6 216 = 108 X 2 224 112 X 2 222 111 X 2 218 = 109 X 2 218

V6 (cont) 109 X 2 550 = 110 X 5 333 = 111 X 3

B

V6 112 327 109 X 3 222 = 111 X 2



I

same in both.

ventricular beats in that discharge of the parasystolic center does not require a triggering impulse Instead the parasystolic pacemaker fires off automatically rhythmically and at its own inherent rate of impulse formation (Fig 27-) Thus the ectopic ventricular *beats follow sinus beats by irregular intervals* (a characteristic often referred to as *inconstant* or *variable* coupling) but bear a fixed relationship to one another from the standpoint of their interectopic intervals In parasystole all interectopic intervals in a given electrocardiogram can be demonstrated to be whole-*number multiples of the shortest* interectopic interval measured or they can all be reduced to a common-denominator interval which corresponds to a single cycle of the ectopic pacemaker This testifies to the striking regularity with which the parasystolic pacemaker forms and discharges impulses even though many of the ectopic impulses fail to elicit a ventricular response The regularity of the parasystolic rhythm indicates that the ectopic pacemaker is "protected in some way against premature discharge by sinus impulses as they spread through the ventricular myocardium The protective mechanism is generally believed to consist of an area of unidirectional entrance *block* surrounding the parasystolic pacemaker al

on two factors (1) the relative rates of the sinus and parasystolic rhythms (parasystolic rhythms are usually slow but rates ranging between 20 and 200 per minute may be seen) and (2) the presence or absence of *exit block* (see below) or its equivalent around the ectopic pacemaker The faster the sinus rhythm and the slower the ectopic rhythm the more likely it is that an ectopic impulse will be discharged while the ventricles are still refractory from previous excitation by a sinus beat In other words the sinus beat interferes with the parasystolic impulse in the ventricles Sometimes the sinus and ectopic impulses arrive more or less simultaneously in the ventricles and each activates a *part of the myocardium* Since two excitation processes jointly produce the ventricular deflection the latter presents a configuration intermediate between that of parasystolic and sinus beats elsewhere in the record This is called a *ventricular fusion beat* and the presence of beats of this type is strongly suggestive of ventricular parasystole Al

though ventricular fusion beats are typical of parasystole they may also be observed when ventricular extrasystoles with long coupling intervals occur during sinus arrhythmia

Exit block surrounding the parasystolic pacemaker is suggested by the following (1) a parasystolic impulse calculated to arrive outside the refractory period of the ventricular myocardium fails to produce a ventricular beat and (2) the calculated rate of the parasystolic pacemaker exceeds that of the sinus node but the expected paroxysmal ectopic tachycardia fails to appear In either of the foregoing circumstances it must be assumed that some of the impulses discharged by the parasystolic center are prevented from entering and activating the ventricular myocardium The specific mechanism responsible for the so called exit block has not been established with certainty

The electrocardiographic features of ventricular parasystole can be summarized as follows

1 The ectopic ventricular beats are not coupled to a preceding beat—that is they do not follow the initial sinus beat by a relatively fixed interval
2 Although the interectopic intervals may differ in length they are all whole number multiples of a single common interval which corresponds to the cycle length of the parasystolic pacemaker
3 The ectopic beats themselves display the widened deformed appearance characteristic of beats arising in the ventricles below the bifurcation of the bundle of His In addition to the conducted sinus beats and parasystolic beats ventricular fusion beats of intermediate contour are usually observed in ventricular parasystole and constitute a finding of diagnostic significance Very infrequently ventricular fusion beats occur with coupled ventricular extrasystoles having long fixed coupling intervals particularly if sinus arrhythmia is also present ventricular fusion beats are sometimes observed in almost complete atrioventricular block with idoventricular pacemaker

The identification of ventricular parasystole has been stressed because ectopic ventricular beats due to parasystole are almost always indicative of underlying cardiac disease while coupled ectopic beats may occur in normal and abnormal hearts (Figs 276–279)

Ectopic Tachycardias; Flutter and Fibrillation

PAROXYSMAL ECTOPIC TACHYCARDIA

Mechanisms

In its most abbreviated form, paroxysmal ectopic tachycardia may consist of as few as six ectopic beats appearing one after another in rapid succession. The first ectopic beat is triggered by the last sinus impulse or by the last beat of the prevailing rhythm, while each subsequent extrasystole is initiated by, and in turn initiates, another ectopic beat. In reality, paroxysmal tachycardia is composed of consecutive coupled

rhythms [illegible]
evidence favoring the close affinity between paroxysmal tachycardia and isolated coupled ectopic beats. In fact, the theories relating to the mechanism of paroxysmal tachycardia, which are summarized below, are merely extensions of those previously described in the section on coupled extrasystoles in Chapter 25.

Re entry.—The theory of re entry proposes that the initial sinus impulse, after exciting all responsive muscle, returns by a devious route to a focus of previous refractoriness. Here the excitation impulse is picked up by the now responsive tissues and introduced back again into conduction pathways traversed in the initial cycle. The re-entry impulse produces an ectopic beat which is coupled to the preceding sinus beat. If, instead of a single re entry cycle, there occur repeated cycles of re entry, it is thought that paroxysmal tachycardia may result.

Single focus of subthreshold activity.—An ectopic focus of automatic impulse formation—that is, a parasystolic pacemaker—on rare occasions may give rise to paroxysmal tachycardia. With this exception, most tachycardias are believed by Scherf to have the same underlying mechanism as coupled ectopic beats, the only difference being that each paroxysmal beat provides the triggering impulse for the next to follow. The ability of the ectopic focus to respond so rapidly and repetitively to initiating stimuli is probably related to the rapidity with which the focus, after discharge, regains its previous subthreshold state.

General Considerations

Paroxysmal tachycardias may arise in the atria, atrioventricular node or ventricles. However, electrocardiographically it is frequently difficult, and sometimes impossible, to differentiate with any degree of certainty an atrial tachycardia from a nodal tachycardia. In this text, the terms *atrial* and *atrioventricular nodal* will not be used when the identity of a tachycardia is uncertain or when the facts under discussion are equally relevant to both types of tachycardia. Instead, paroxysmal tachycardias arising in the atrioventricular node or above will be referred to as *supraventricular tachycardias* and discussed under this general heading. In fact, there is little practical advantage to be gained from the differentiation of atrial and nodal tachycardias, since their clinical implications, therapy and, as already mentioned, electrocardiographic features are often similar. On the other hand, clinically and in other respects, ventricular tachycardias are entirely different from supraventricular tachycardias. In short, the salient electrocardiographic fact to be established with reference to a tachycardia is its site of origin, whether supraven

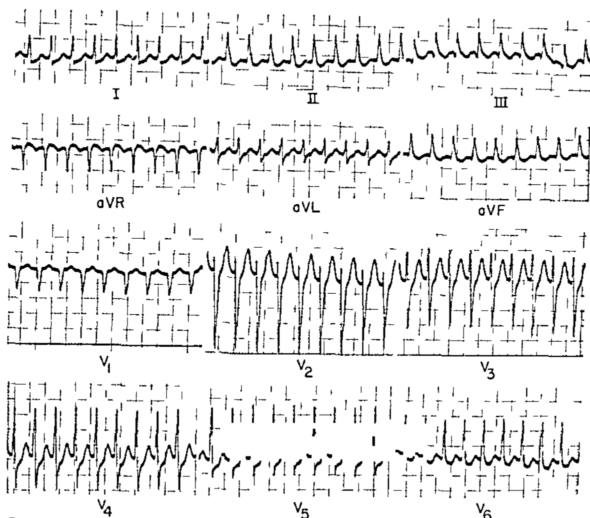


Fig 280 –*Paroxysmal supraventricular tachycardia probably originating in the atria The ventricular rate is 215* beats per minute
QRS deflections
cardia The depre

tricular (atrial or atrioventricular nodal) or ventricular

Atrial nodal and ventricular tachycardias have the following features in common

1 The ectopic tachycardias occur in paroxysms of abrupt onset and offset

2 The paroxysms tend to recur the likelihood of such a recurrence being all the greater if there is a history of any previous attacks

3 Before after and during intervals between at tacks of paroxysmal tachycardia the electrocardio gram frequently records single ectopic beats resem bling those present during tachycardia and having the same coupling interval as the first beat of a paroxysm of tachycardia

4 Occasionally paroxysmal tachycardia persists only for a matter of minutes and passes without no tice Tachycardias which produce symptoms usually last several hours but may continue without interrup tion for days or rarely weeks Sometimes paroxysmal tachycardia takes the form of a *repetitive tachycardia* which is characterized by persistently recurring par oxysms of approximately 20–200 extrasystoles each paroxysm separated from the other by several sinus beats These short interrupted runs of tachycardia tend to be repeated over and over again and in gen eral are relatively refractory to therapy Repetitive *tachycardia may continue for months and then sub* side spontaneously never to reappear

5 Offset of a paroxysmal tachycardia is often fol lowed by a long pause before the sinus pacemaker finally takes over or there may be a period of slow sinus rhythm or of nodal escape The reason for this is that the ectopic tachycardia depresses the sinus node In a badly diseased heart this depression can be of such severe degree as to lead to prolonged cardiac standstill

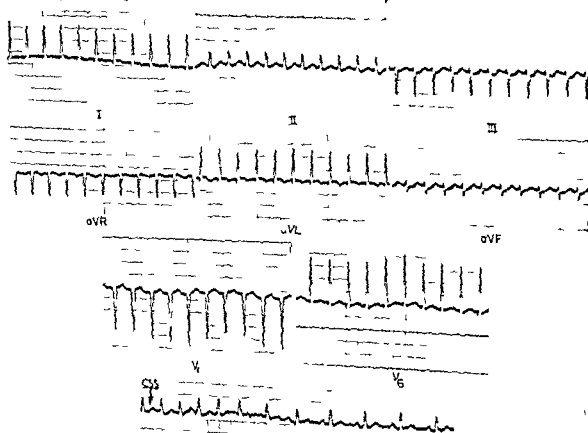
6 After a prolonged episode of paroxysmal tachy

cardia older patients with cardiac disease and occasionally some persons without detectable heart disease may show abnormal T wave changes in their electrocardiograms The findings can persist from several hours or days to several weeks after termination of the tachycardia This has been designated the *post tachycardia electrocardiographic syndrome* Several fatal cases of this type were found at autopsy to have diffuse areas of subendocardial fibrosis

Paroxysmal Supraventricular Tachycardia

In paroxysmal supraventricular tachycardia, the P waves when identifiable usually differ from those observed in the same patient during sinus rhythm In tachycardias originating in the cephalic region of the atria the ectopic P waves are upright in leads I and II Tachycardias arising in the caudal aspect of the atria or in the atrioventricular node are associated with inverted P waves in leads II III and aVF and upright P w[illegible] aVR In both atrial and nodal tachycardia [illegible] ally of nor[illegible] from the same patient during sinus rhythm [illegible] 250) The exception to this rule is that ventricular aberration sometimes may alter the appearance of the QRS complexes in either type of tachycardia as will be described later Occasionally nodal and atrial tachycardias can be differentiated from each other by the fact that in nodal tachycardia inverted P waves precede the QRS complexes by less than 0 12 second while in atrial tachycardia upright or inverted P waves are followed by ventricular complexes after a P–R interval of 0 12 second or longer However it is often difficult to be certain whether the inverted P waves are associated with the preceding or following

Fig 28
proximatel
lead strip
momentary [illegible]



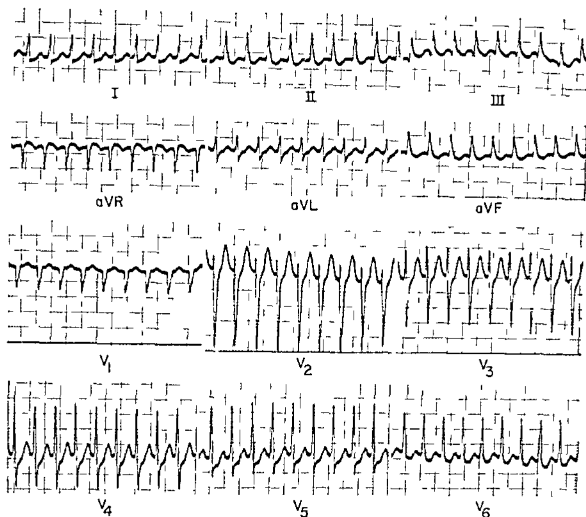


Fig 280 —Paroxysma
beats per minute Atrial
QRS deflections are indi
cardia The depressed S–

tricular (atrial or atrioventricular nodal) or ventricular

Atrial nodal and ventricular tachycardias have the following features in common

1 The ectopic tachycardias occur in paroxysms of abrupt onset and offset

2 The paroxysms tend to recur the likelihood of such a recurrence being all the greater if there is a *history of any previous attacks*

3 Before after and during intervals between attacks of paroxysmal tachycardia the electrocardiogram frequently records single ectopic beats resembling those present during tachycardia and having the same coupling interval as the first beat of a paroxysm of tachycardia

4 Occasionally paroxysmal tachycardia persists only for a matter of minutes and passes without notice Tachycardias which produce symptoms usually last several hours but may continue without interruption for days or rarely weeks Sometimes paroxysmal tachycardia takes the form of a *repetitive tachycardia* which is characterized by persistently recurring paroxysms of approximately 20–200 extrasystoles each paroxysm separated from the other by several sinus beats These short interrupted runs of tachycardia tend to be repeated over and over again and in general are relatively refractory to therapy Repetitive tachycardia may continue for months and then subside spontaneously never to reappear

5 Offset of a paroxysmal tachycardia is often fol*lowed by a long pause before the sinus pacemaker* finally takes over or there may be a period of slow sinus rhythm or of nodal escape The reason for this is that the ectopic tachycardia depresses the sinus node In a badly diseased heart this depression can be of such severe degree as to lead to prolonged cardiac standstill

6 *After a prolonged episode of paroxysmal tachy*

during sinus rhythm and their durat on does not exceed 0 10 second Howe er if the rate of the tachycardia is sufficiently rapid, the intraventricular conducting pathways and ventricular muscle may not have enough time to recover completely before as r ... e ... succeeding impulse This can lead to

conduction disturbance is present during an atrial tachycardia, the abnormal appearance of the ventricular complexes may give the impression that the tachycardia originates in the ventricles particularly if the ectopic P waves of the atrial tachycardia are poorly defined or obscured Atrioventricular nodal tachycardia with ventricular aberra..on cannot ordinarily be differentiated from ventricular tachycardia unless the recorded onset or offset of the paroxysm demonstrates frequent nodal extrasystoles

Whenever the electrocardiogram shows a tachycardia of apparent supraventricular origin an alternative diagnostic possibility—which should always be entertained and, if possible excluded—is atrial flutter with per sting 2 1 atrioventricular response Carotid sinus stimulation should always be attempted in such a situation, since the resulting vagal stimulation causes the ventricular rate to slow in atrial flutter whereas it either abolishes or has no effect on a supraventricular tachycardia (Figs 281 and 282) The differential diagnosis of atrial flutter and supraventricular tachycardia is considered at greater length later in this chapter

In the experience of some investigators, paroxysmal supraventricular tachycardias with very rapid rates seem less likely to be terminated by carotid sinus stimulation than those with slower rates Although supraventricular tachycardia typically shows 1 1 atrioventricular response variations in atrioventricular conduction may occur running the gamut from interference phenomena such as P R interval prolongation or 2 1 atrioventricular response (Fig 283) to high degrees of atrioventricular block In tachycardias with very rapid rates diastole may be so shortened that the junctional tissues are never able to recover completely This may lead to slower atrioventricular conduction of the supraventricular impulses or to 2 1 atrioventricular conduction in which every other impulse arrives at the atrioventricular node during its normal refractory period and is not conducted In either case one supraventricular impulse interferes with atrioventricular conduction of the following impulse (atrioventricular interference) Failure of every second impulse to be conducted may occur in tachycardias with relatively

... refractory period of the atrioven...

terference or atrioventricular block is sometimes impossible to determine but in general 2 1 atrioventricular response in tachycardias with rates below 200 beats per minute implies depressed atrioventricular conductivity On the other hand the atrioventricular junctional tissues cannot normally maintain a 1 1 response to more than 200-250 impulses per minute and so 2 1 conduction in tachycardias within this rate can be attributed to atrioventricular interference

Three-to-one (or 4 1, 5 1 etc) atrioventricular response or variable atrioventricular response in supraventricular tachycardia is usually indicative of impaired atrioventricular conductivity and may be produced by excess digitalis by degenerative or in...

is not necessarily an indication of atrioventricular block in this instance since the nonconducted P waves arrive at ...

always falls within the Q-T interval of the preceding ventricular beat.

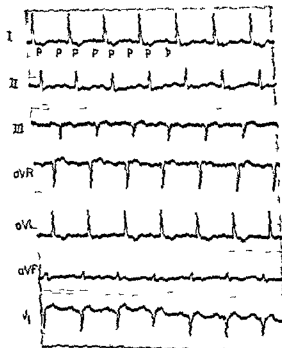


Fig 283 —Paroxysmal atrial tachycardia with 2 1 atrioventricular response The rate of the paroxysmal atrial tachycardia is 200 beats per minute while the ven...

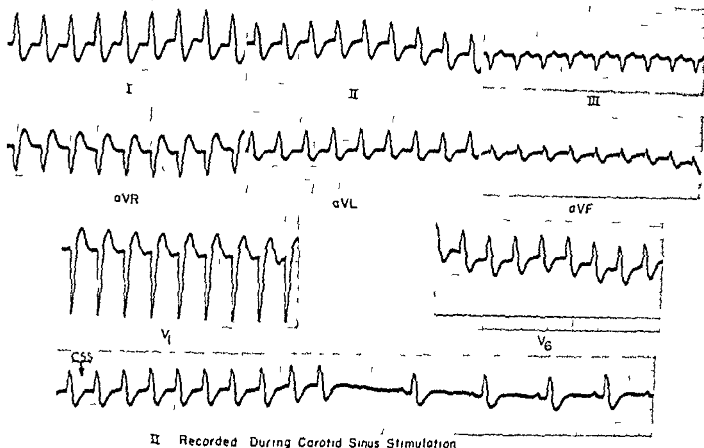


Fig 282 —Paroxysmal tachycardia with a ventricular rate of about 150 beats per minute The ventricular rhythm is

QRS complexes and so in many instances the P–QRS relationship is not helpful in separating the two types of supraventricular tachycardia When present atrioventricular dissociation is perhaps the most reliable point of distinction between an atrial and an atrioventricular nodal tachycardia provided of course the two do not coexist (combined paroxysmal tachycardias) Even though atrioventricular dissociation is present the diagnosis of atrioventricular nodal tachycardia can only be made if the ventricular deflections have a duration of 0 10 second or less If the QRS interval is wider than this it usually is impossible to differentiate nodal tachycardia with aberration from ventricular tachycardia

Paroxysmal supraventricular tachycardias can exhibit rates ranging from a little over 100 beats per minute in nodal tachycardias or 140 beats per minute in atrial tachycardias to as fast as 220 beats per minute, although rates of 150–190 beats per minute are commonly encountered Once a supraventricular tachycardia has become established its rate remains constant and its rhythm strikingly regular the cycle length varying only infrequently by more than 0 01 second from beat to beat However during onset of a paroxysm the ectopic beats occur somewhat irregularly and with an accelerating rate until finally the tachycardia becomes established This initial phase is referred to as the *warm up* period of the tachycardia Shortly before offset of a tachycardia there is slowing of its rate and with the appearance of the last beat of the paroxysm a noncompensatory pause follows Whether the first postectopic beat proves to be a sinus beat a supraventricular beat from a displaced pacemaker or an atrioventricular nodal or ventricular escape beat is determined largely by the degree of sinus node depression induced by the supraventricular tachycardia as well as by the relative rhythmicity of the ectopic pacemakers

In the usual supraventricular tachycardia the configuration of the QRS complexes is the same as

during sinus rhythm and their duration does not exceed 0 10 second However if the rate of the tachycardia is *sufficiently rapid* the intraventricular conducting pathways and ventricular *muscle may not* have enough time to recover completely before arrival of each succeeding *impulse* This can lead to aberrant intraventricular *conduction* of the supraventricular impulses or to the *appearance* of previously latent bundle branch block When an intraventricular conduction disturbance *is present* during an atrial tachycardia, the abnormal appearance of the ventricular complexes may give the impression that the tachycardia originates in the *ventricles particularly* if the ectopic P waves of the atrial tachycardia are poorly defined or obscured. *Atrioventricular nodal* tachycardia with ventricular aberration cannot ordinarily be differentiated from *ventricular tachycardia* unless the recorded onset *or offset* of the paroxysm demonstrates frequent nodal extrasystoles

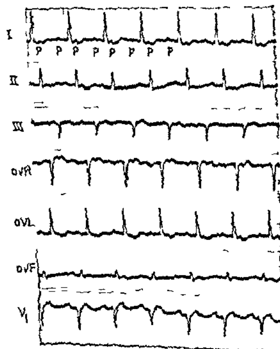
Whenever the electrocardiogram shows a tachycardia of apparent supraventricular origin an *alternative* diagnostic possibility—which should always be entertained and if possible excluded—is atrial flutter with persisting 2 1 atrioventricular *response* Carotid sinus stimulation should always be *attempted* in such a situation, since the resulting vagal *stimulation* causes the ventricular rate to slow in atrial flutter whereas it either abolishes or has no *effect on a supraventricular* tachycardia (Figs 281 and 282) The differential diagnosis of atrial flutter and *supraventricular tachycardia* is considered at greater length later in this chapter

In the experience of some investigators paroxy*smal* supraventricular tachycardias with very *rapid rates* seem less likely to be terminated by carotid sinus stimulation than those with slower rates Although supraventricular tachycardia *typically shows 1 1* atrioventricular response variations in atrioventricular conduction may occur running the gamut from interference phenomena such as P–R interval prolongation or 2 1 atrioventricular response (Fig 283) to high degrees of atrioventricular block In tachycardias with very rapid rates diastole may be so shortened that the junctional tissues are never able to recover completely This may lead to slower atrioventricular conduction of the supraventricular impulses or to 2 1 atrioventricular conduction in which every other impulse arrives at the atrioventricular node during *its normal* refractory period and is not conducted. In either case one supraventricular impulse interferes with atrioventricular conduction of the following impulse (atrioventricular interference) Failure of every second impulse to be conducted *may* occur in tachycardias with relatively slower rates if the refractory period of the atrioventricular node is prolonged (atrioventricular block) The mechanism involved, whether atrioventricular interference or atrioventricular block is sometimes impossible to determine *but in general* 2 1 atrioventricular response *in tachycardias* with rates below 200 beats per minute implies depressed atrioventricular conductivity On the other hand the atrioventricular junctional tissues cannot normally maintain a 1 1 response to more than 200–250 impulses per minute and so 2 1 conduction in tachycardias within this rate can be attributed to atrioventricular interference

Three to-one (or 4 1 5 1 etc) atrioventricular response or variable atrioventricular response *in supraventricular* tachycardia is usually *indicative of* impaired atrioventricular conductivity and may be produced by excess digitalis by degenerative or in

Fig 283 —Paroxysmal atrial tachycardia *with* 2 1 *atrioventricular* response The rate of the paroxysmal atrial tachycardia is 200 beats per minute while the ventricular rate is exactly one half as fast. The symbol P in

this instance since *the nonconducted* P waves arrive at the atrioventricular junction during its *normal refractory period It will be* recalled that the normal refractory period of the atrioventricular node *coincides approximately* with the Q–T interval of the electrocardiogram. In the tachycardia record shown here the nonconducted P wave always falls within the Q–T interval of the preceding ventricular beat

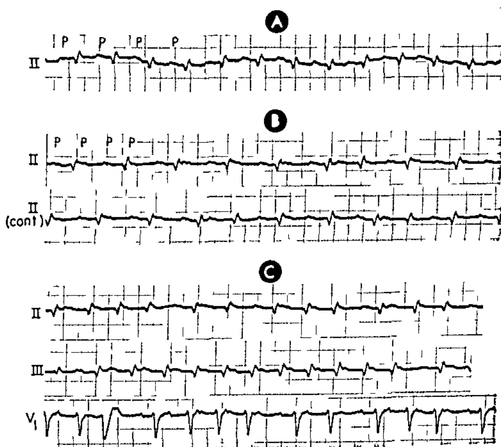


flammatory changes in the atrioventricular node or by other influences Sometimes when the refractory period of the atrioventricular node is lengthened by one of the foregoing factors recovery of the depressed junctional tissues after each conducted beat may be progressively less complete Successive supraventricular beats are conducted therefore with gradually lengthening P–R intervals until one beat finally arrives during the absolute refractory period of the node and is blocked (Fig 284) Because of the longer recovery period allotted the atrioventricular node by the resulting pause the first conducted beat thereafter has a shorter P–R interval than those following which show the same progressive increments in their P–R intervals as the beats of the preceding cycle This cyclic change in the P–R intervals of successive beats called the Wenckebach phenomenon typifies the common form of incomplete atrioventricular block (Figs 285–287)

In addition to variations in forward atrioventricular conduction such as those just described (Fig 288) nodal tachycardias frequently are accompanied by disturbances of retrograde conduction (Fig 289) which may present electrocardiographically in the following forms

Atrioventricular dissociation —Failure of the nodal pacemaker to take over the atria can be due to retrograde atrioventricular block and/or interference (Fig 290)

Incomplete retrograde atrioventricular block with Wenckebach phenomenon —This form (see Fig 248) is characterized by a shift of each successive retrograde P wave farther behind the QRS complex of the nodal beat until a retrograde P wave eventually



F[illegible] 28[illegible] [illegible] varying atrioventricular response In A lead II displays a sinus tachy [illegible]

[illegible] ence of varying ratios of atrioventricular response accompanied by [illegible]

Fig 285 —Paroxysmal atrial tachycardia with Wenckebach incomplete atrioventricular block and intermittent left bundle branch block In lead V in A sinus P waves (*P*) appear at a rate of about 60 per minute and there is 3 2 Wenckebach incomplete atrioventricular block Note that the QRS complex of the first conducted sinus beat after the blocked sinus beat is of normal duration while the next conducted sinus beat produces a widened rS deflection sugges tive of complete left bundle branch block Record B was made 1 day after record A P waves (*P*) now appear at a rate of 160 per minute and their configuration in lead V differs from that in the same lead in A thus a paroxysmal atrial

it lead strips in

uperimposed on

–R intervals of

[illegible] during [illegible] atrioventricular conduction show gradual lengthening and that the [illegible]

[illegible] decreases)

flammatory changes in the atrioventricular node or by other influences Sometimes when the refractory period of the atrioventricular node is lengthened by one of the foregoing factors recovery of the depressed junctional tissues after each conducted beat may be progressively less complete Successive supraventricular beats are conducted therefore with gradually lengthening P–R intervals until one beat finally arrives during the absolute refractory period of the node and is blocked (Fig 284) Because of the longer recovery period allotted the atrioventricular node by the resulting pause the first conducted beat thereafter has a shorter P–R interval than those following which show the same progressive increments in their P–R intervals as the beats of the preceding cycle This cyclic change in the P–R intervals of successive beats called the Wenckebach phenomenon typifies the common form of incomplete atrioventricular block (Figs 285–287)

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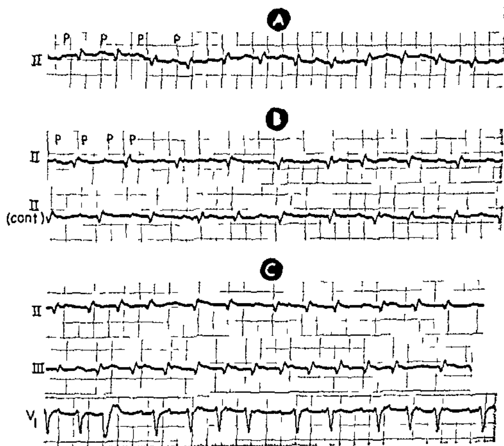


Fig 284 —Paroxysmal atrial tachycardia with varying atrioventricular response In **A** lead II displays a sinus tachycardia with each atrial P wave (*P*) being conducted into the ventricles However in **B** the continuous strip of lead II bach incomplete atrioventricular block In **C** there is 4 3 Wenckebach atrioventricular conduction and at other times 1 1 atrioventricular conduction The presence of varying ratios of atrioventricular response accompanied by the Wenckebach phenomenon is usually indicative of an abnormal refractoriness of the atrioventricular junctional tissues whether due to intrinsic atrioventricular node disease or to depression secondary to digitalis effect or vagal stimulation



F ... P ... mal atrioventricular nodal tachycardia with retrograde activation of the atria and a ventricular rate ... ventricular complex by an R–P interval

fails to appear or is replaced by a sinus P wave

Incomplete retrograde atrioventricular block with constant R–P intervals –This type (Fig 291) usually produces 1 2 atrioventricular response every other retrograde P wave being absent Sometimes the dropping of a retrograde P wave occurs at irregular intervals

First degree retrograde block as evidenced by R–P intervals of 0 20 second or longer undoubtedly occurs in nodal tachycardia but is usually impossible to establish with certainty because the lengthened R–P interval makes it all the more difficult to determine whether the retrograde P wave accompanies the preceding or the following ventricular complex

The frequency with which paroxysmal atrial tachycardia with atrioventricular block occurs as a manifestation of digitalis intoxication has been emphasized by many investigators This form of supraventricular tachycardia is thought to resemble physiologically atrial flutter because of (*a*) the manner in which the paroxysmal atrial tachycardia with atrioventricular block responds to various therapeutic agents and (*b*) its *refractoriness in most* instances to carotid sinus stimulation Therefore it is not surprising that the differentiation of atrial tachycardia with atrioventricular block from a relatively slow atrial flutter often presents a difficult problem As a general rule an atrial rate above 200 beats per minute favors atrial flutter while a slower atrial rate supports the diagnosis of atrial tachycardia Other equally arbitrary

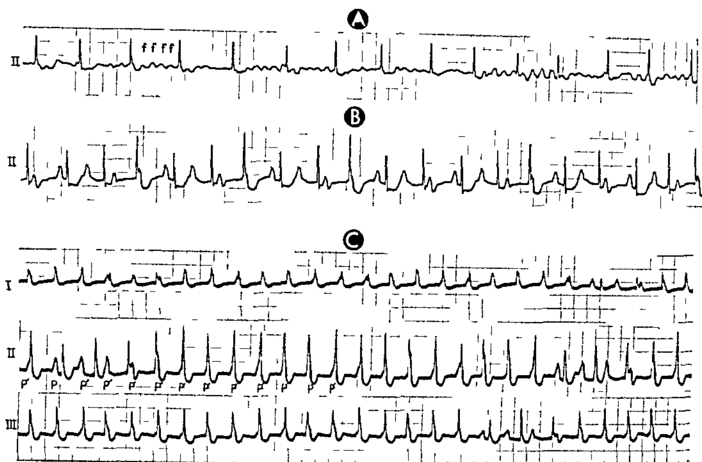


Fig 286 —Transition from atrial fibrillation to paroxysmal atrial tachycardia with atrioventricular block occurring
s were recorded from the same patient during the

complete atrioventricular block with varying ratios of atrioventricular response Consequently the strip of lead II in **B**
with atrioventricular block (This lead strip was recorded
strips in **C** were obtained about 1 hour later than those in **B**
me rate as previously and the ectopic P waves (P) are best
demonstrated in lead II The P wave buried in the first QRS complex in lead II is nonconducted while the next P wave
-R intervals
Finally the
again Thus
for the greater portion of the lead strip there is 1 1 atrioventricular conduction

points of distinction have been proposed but none of the present criteria are entirely satisfactory According to Lown Wyatt and Levine the electrocardiographic features which tend to distinguish paroxysmal atrial tachycardia with atrioventricular block from atrial flutter are as follows

1 In atrial flutter the atrial rate generally exceeds 250 beats per minute while in paroxysmal atrial tachycardia with atrioventricular block the atrial rate usually ranges from 150 to 250 beats per minute.

2 In atrial flutter the atrial deflections in leads II and III are downwardly directed and are associated with an oscillating saw tooth appearance of the base line In paroxysmal atrial tachycardia with block the atrial deflections are upright in leads II and III and are separated by an isoelectric base line (Fig 283)

3 In pure atrial flutter the atrial rhythm is perfectly regular but in about one half of the cases of paroxysmal atrial tachycardia with block the P–P intervals vary in length by 0 02–0 12 second

Clinical Significance of Supraventricular Tachycardia

Supr

sociates there was a 3 6% incidence of sup lar tachycardia Of the patients with electrocardiograms showing supraventricular tachycardia about one third were considered to have normal hearts another third had rheumatic heart disease and about one sixth arteriosclerotic heart disease The remaining patients had thyrocardiac or hypertensive heart disease or other conditions The association of supraventricular tachycardia with the Wolff Parkinson White syndrome and various forms of congenital heart disease such as interatrial septal defect and Eisenmenger s complex is also well recognized

A person with a normal heart usually tolerates supraventricular tachycardia reasonably well unless the ventricular rate is very fast Inasmuch as the car



F ng n ... nodal tachycardia with retrograde activation of the atria and a ventricular rat

l w the QRS deflections Moreover in lead aVF comparison of the QRS configuration of the first several sinus bea

verted sinus P waves (P) The reason for the variation in retrograde atrioventricular conduction of the atrioventricul nodal impulses in the above record is not clear

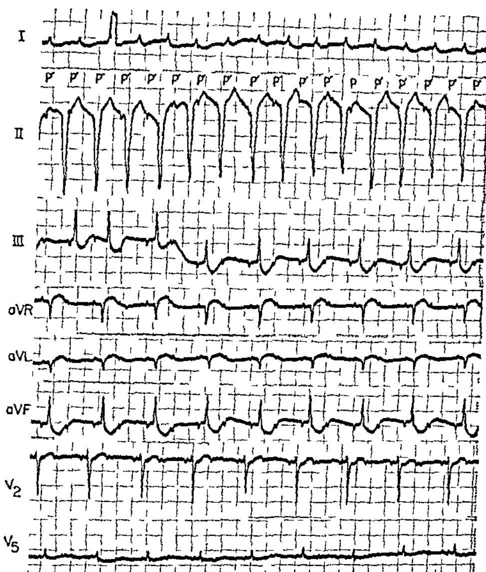


Fig 288 —The mechanism and site of origin of the ectopic tachycardia present in this record cannot be established
145 beats per min
and are inverted in
ts occurring in this
I ... tion in the P–R intervals suggests that there is dissociation of the atrial and ventricular rhythms Despite the aberration of the QRS deflections in leads I and II their normal duration tends to exclude the possibility of ventricular tachycardia and favors tachycardia of atrioventricular nodal origin One possible explanation for the findings in lead I and II is that the inverted P waves in lead II represent an atrial tachycardia originating low in the atria while an atrioventricular nodal tachycardia is simultaneously present in the ventricle with incomplete or complete atrioventricular dissociation In lead III the first inverted P wave is not conducted while the second and third P waves are transmitted into the ventricles with gradually lengthening P–R intervals The fourth P wave is not conducted and from that point on throughout the remaining portion of lead III and the rest of the lead strips shown there is supraventricular tachycardia with 2 1 atrioventricular conduction If the findings in lead I and II were to be ignored then the remaining leads of the electrocardiogram could be interpreted alternatively as atrioventricular nodal tachycardia or as atrial tachycardia with antegrade 2 1 atrioventricular block

points of distinction have been proposed but none of the present criteria are entirely satisfactory According to Lown Wyatt and Levine the electrocardiographic features which tend to distinguish paroxysmal atrial tachycardia with atrioventricular block from atrial flutter are as follows

1 In atrial flutter the atrial rate generally exceeds 250 beats per minute while in paroxysmal atrial tachycardia with atrioventricular block the atrial rate usually ranges from 150 to 250 beats per minute.

2 In atrial flutter the atrial deflections in leads II and III are downwardly directed and are associated with an oscillating saw tooth appearance of the base line. In paroxysmal atrial tachycardia with block, the atrial deflections are upright in leads II and III and are separated by an isoelectric base line (Fig 283)

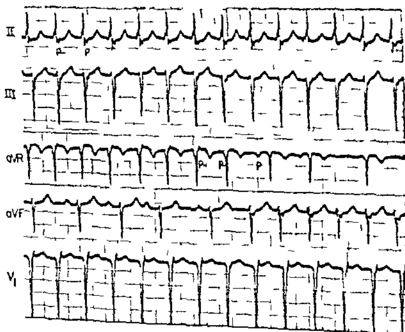
3 In pure atrial flutter the atrial rhythm is perfectly regular but in about one half of the cases of paroxysmal atrial tachycardia with block, the P–P intervals vary in length by 0 02–0 12 second.

Clinical Significance of Supraventricular Tachycardia

Supraventricular tachycardias like single coupled ... may occur in normal or

sociates there was a 3 6% incidence of supra... lar tachycardia. Of the patients with electrocardiograms showing supraventricular tachycardia about one third were considered to have normal hearts an other third had rheumatic heart disease and about one sixth arteriosclerotic heart disease The remaining patients had thyrocardiac or hypertensive heart disease or other conditions The association of supraventricular tachycardia with the Wolff Parkinson White syndrome and various forms of congenital heart disease such as interatrial septal defect and Eisenmenger s complex, is also well recognized

A person with a normal heart usually tolerates supraventricular tachycardia reasonably well unless the ventricular rate is very fast Inasmuch as the car



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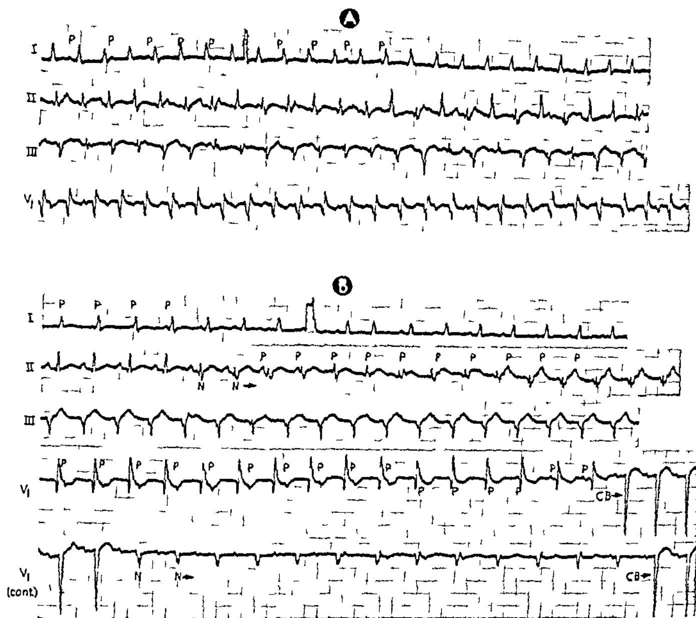


Fig 290 —Paroxysmal atrioventricular nodal tachycardia with atrioventricular dissociation The lead strips in **A** display a relatively regular tachycardia with a ventricular rate of about 150 beats per minute and QRS deflections of relatively normal duration With some difficulty upright sinus P waves (*P*) can be identified in all four lead strips shown the P waves occurring at a slower rate and with a rhythm unrelated to the ventricular rhythm In view of the latter findings the diagnosis of atrioventricular dissociation (with sinoatrial rhythm in the atria) can be made and the ventricu

e of the normal duration of the

on within a given lead strip may

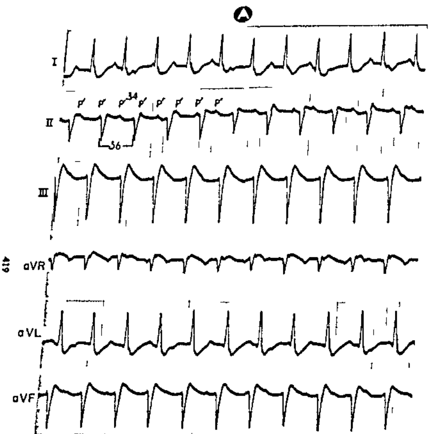
nodal beats to display ventricu

lar aberration The lead strips presented in **B** were recorded from the same patient a short time after those in **A**. The mechanism of the rhythm is best illustrated in leads II and V_1 The first four ventricular beats in lead II are produced by conducted sinus impulses however the fifth and subsequent ventricular complexes (*N*) are of atrioventricular nodal origin and occur at a rate of about 112 beats per minute Sinus P waves (*P*) appearing at a slightly slower rate can be spaced off without interruption indicating that there is atrioventricular dissociation Note that, with onset of the atrioventricular nodal rhythm there is an immediate change in QRS configuration although there is only slight prolongation of the QRS duration In lead V_1 there is a long period of

dissociation which at the point indicated by the arrow and *CB*

ance of sinus rhythm in the ventricles Note that with onset of

nodal tachycardia differs from that of the nodal beats present in the atrioventricular nodal tachycardia occurring in the first half of the lead strip of lead V



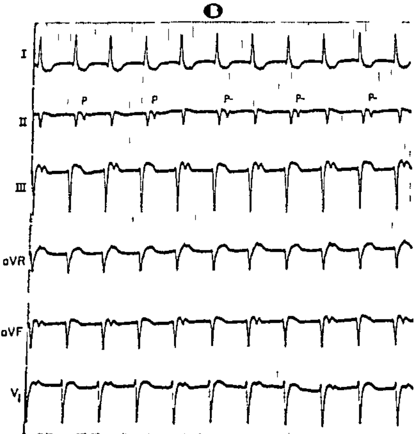


Fig 291 —In **A** there are present simultaneously a paroxysmal atrial tachycardia (atrial beats are labeled *P′*) with an atrial rate of about 170–180 beats per minute and an atrioventricular nodal tachycardia with a ventricular rate of 105 beats per minute The atrial tachycardia is completely unrelated to the atrioventricular nodal tachycardia and so a diagnosis of combined or double paroxysmal supraventricular tachycardia with atrioventricular dissociation can be made In this example the double tachycardia is probably a manifestation of excess digitalis effect Record **B** was obtained from the same patient several hours later following treatment with potassium chloride Atrioventricular nodal tachycardia persists but exhibits a slower rate than was present in **A** However the P waves which were upright in **A** and appeared at a rate and rhythm different from the ventricular deflections are inverted in leads II III and aVF of **B** therefore they are retrograde P waves (*P*−) In addition the inverted P waves follow every second ventricular beat and so the mechanism of the rhythm in the electrocardiogram in **B** is atrioventricular nodal tachycardia with 1 2 retrograde atrioventricular block

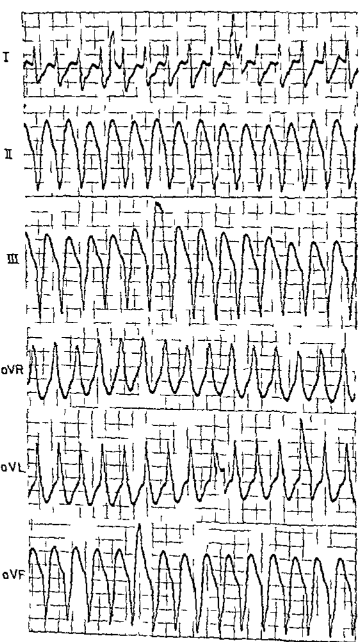


Fig 292.—A paroxysmal ventricular tachycardia with a ventricular rate of about 158 beats per minute. The ventricular complexes are bizarre in appearance and prolonged in duration. In the absence of evidence to the contrary these features must be considered indicative of ventricular as opposed to supraventricular tachycardia. Atrial deflections cannot be identified. (*Continued.*)

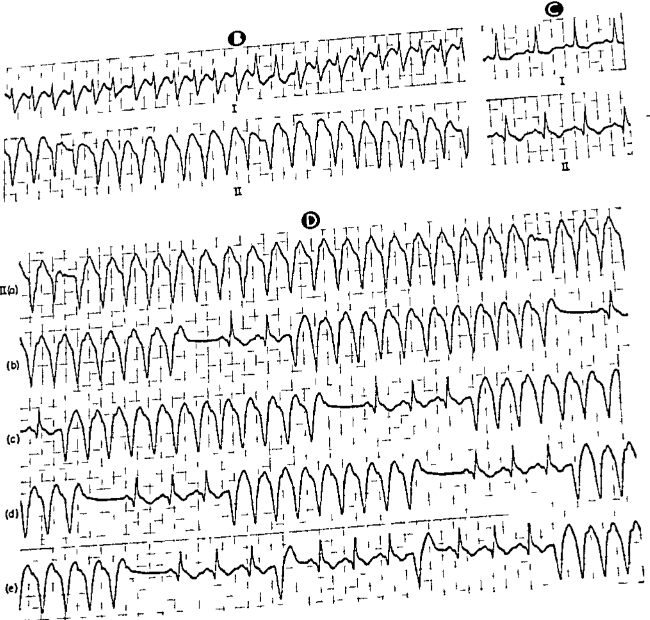


Fig 292 (*cont*) —**B** strips of leads I and II showing short runs of ventricular tachycardia separated by QRS deflections of different configuration which probably represent conducted sinus beats **C** sinus rhythm Note the marked difference in the QRS configuration during sinus rhythm as compared to that previously noted during ventricular tachycardia **D** five lead strip (continuous strips of lead II) recorded from the same patient as the preceding and demonstrating short recurring paroxysms of ventricular tachycardia separated by conducted sinus beats This phase of the ventricular tachycardia displays some of the characteristics of a *repetitive paroxysmal tachycardia* (see Fig 293)

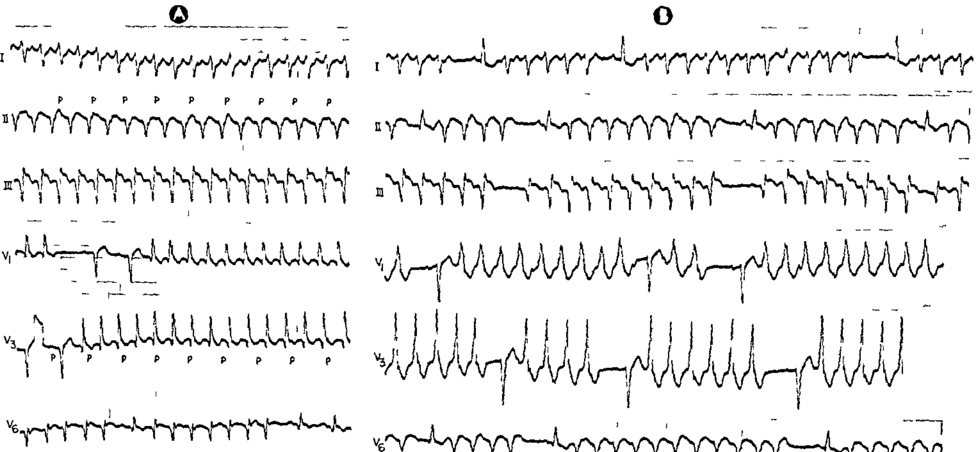


Fig 293 —Repetitive paroxysmal ventricular tachycardia with atrioventricular dissociation. In **A** there is ventricular tachycardia with a rate of about 170 beats per minute which continues without interruption throughout leads I, II and III. Although difficult to identify, P waves (*P*) can nevertheless be spaced off independent of the ventricular rhythm so that there is complete atrioventricular dissociation of the sinoatrial rhythm in the atria and of the ectopic ventricular rhythm in the ventricles. In leads V_1, V and V the ventricular tachycardia begins to take on the characteristics of repetitive tachycardia in that one or two conducted sinus beats are inserted between paroxysms of tachycardia. In **B** the electrocardiographic leads recorded somewhat later from the same patient display the characteristic features of a repetitive ventricular tachycardia. Thus in each lead one can see short bouts of rapid ventricular beats which are separated from one another by one or two conducted sinus beats with a completely different QRS configuration. There is atrioventricular dissociation during each paroxysm of tachycardia.

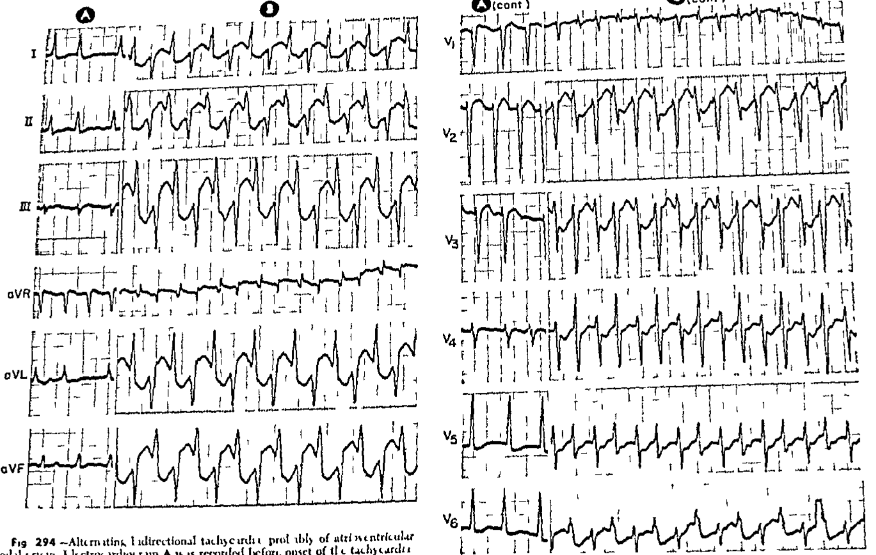


Fig 294 —Alternating bidirectional tachycardia probably of atrioventricular nodal origin Electrocardiogram **A** was recorded before onset of the tachycardia while the mechanism of the rhythm was atrial fibrillation Record **B** was made at the height of the episode of tachycardia Note that not all of the leads record ventricular deflections which alternate in direction In leads V and V in particular the QRS deflections have much the same direction but certainly alternate in appearance Note also that the QRS duration is 0 10 second The intervals between the upright ventricular beats equal the intervals between the downwardly directed ventricular beats However the interval extending from the beginning of an R wave to the beginning of the next QS deflection is longer than the interval extending from the beginning of the QS deflection to the following R wave (Continued)

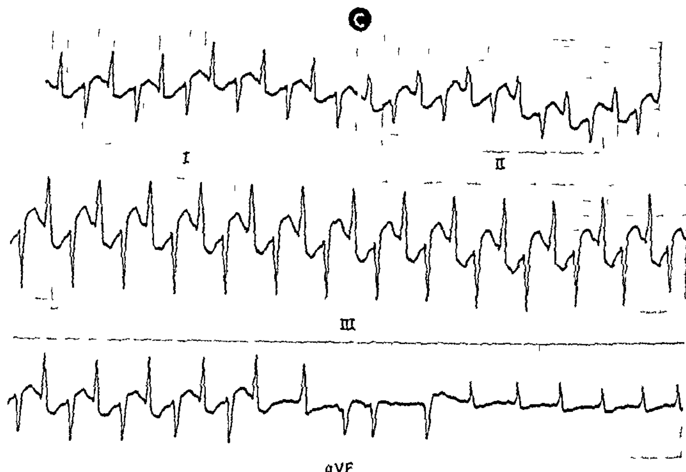


Fig 294 (*cont*) —*The beats appearing in* **C** *were recorded after the patient had received intravenously 0 8 Gm of potassium chloride in an infusion The termination of the bidirectional tachycardia was recorded in the final lead strip of lead aVF the rhythm thereafter being atrial fibrillation with varying atrioventricular conduction*

diac output of normal hearts drops somewhat when the cardiac rate exceeds 180 beats per minute vascular collapse occasionally may be experienced even by normal persons with such rapid rates Fortunately at least half the cases of supraventricular tachycardia occurring in normal hearts either subside spontaneously or can be terminated easily by such simple measures as the Valsalva procedure carotid sinus stimulation vomiting etc In older subjects and particularly in patients with heart disease paroxysms of supraventricular tachycardia displaying only moderately rapid ventricular rates (sometimes even less than 180 per minute) can effect a fall in cardiac output and coronary blood flow Myocardial infarction angina pectoris or acute heart failure may rapidly ensue if the tachycardia is not soon abolished

The theory of paroxysmal supraventricular tachycardia is discussed later

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Fig 295 —*The lead strips in* **A** *show ventricular tachycardia approaching ventricular flutter In the two strips of lead II in* **B** *recorded later (all strips in this figure from the same patient) there appears a typical ventricular tachycardia which does not resemble ventricular flutter as did the previous tracing Lead II (b) was recorded after lead II (a) and shows a change in the QRS configuration of the ventricular beats For the first time atrial deflections are definitely recognizable The atrial waves appear at a rate and rhythm which are independent of the ventricular tachycardia In* **C** *ventricular tachycardia is still present but the rate of the independent atrial rhythm (250 beats per minute) is consistent with the diagnosis of atrial flutter and complete atrioventricular dissociation The presence of flutter is established in* **D** *where the ventricular tachycardia has disappeared and there is predominantly atrial flutter (F flutter waves) with 2 1 atrioventricular response The final lead strip of lead II in* **E** *demonstrates a sinus rhythm with frequent atrial extrasystoles (A) and either occasional atrioventricular nodal extrasystoles (V) with prolonged P–R intervals or atrial extrasystoles originating low in the atria*

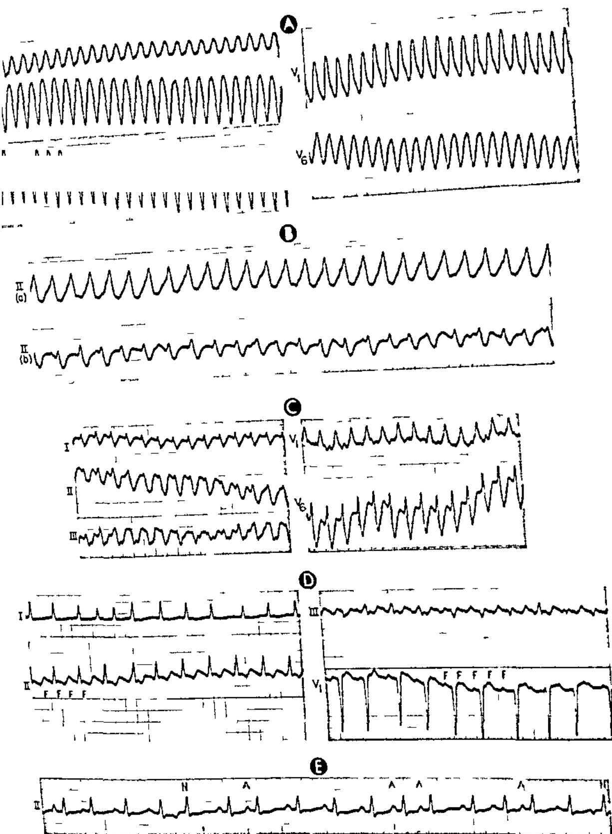


Fig 295 – Legend on facing page.

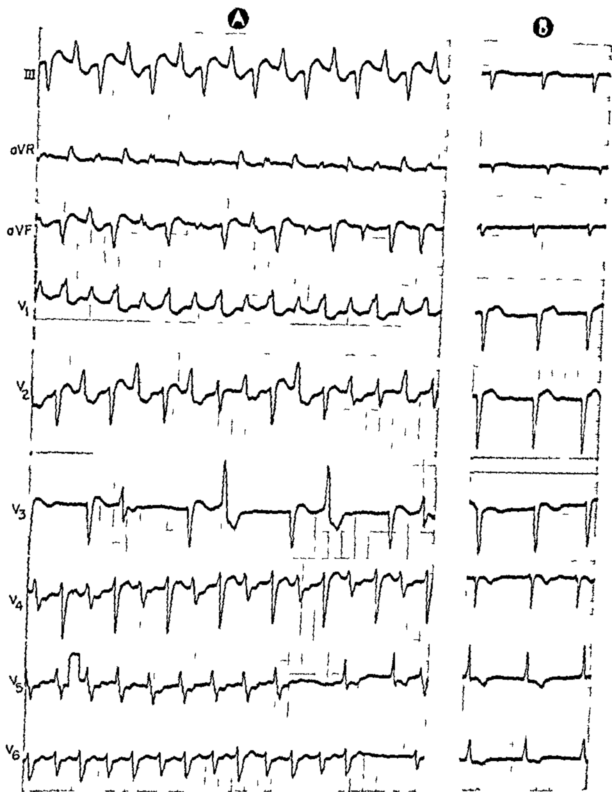


Fig 296 —Alternating bidirectional tachycardia possibly of atrioventricular nodal origin In **A** the electrocardiogram shows the characteristic features of an alternating bidirectional tachycardia but differs from that described in Figure 294 in that the QRS duration of the ventricular beats is 0 12 second The record in **B** was made before onset of the bidirectional tachycardia when the rhythm was atrial fibrillation This record is presented simply to provide a basis for comparison of the QRS configuration present in the tachycardia and that present during fibrillation *(Continued)*

Paroxysmal Ventricular Tachycardia

... ntricular tachycardia consists of

...

... tricular tachycardia are widened (0 1... second or more) and deformed and appear at rates of 130–180 beats per minute (Fig 292) However faster or slower rates may be encountered in ventricular tachycardia More often than not, there is some irregularity of the ventricular rhythm but in many instances the variations in cycle length from beat to beat are no greater than those observed in supraventricular tachycardia (In a typical supraventricular tachycardia the R-R intervals vary by 0 01 second or less)

Once established a paroxysm of ventricular tachycardia may continue uninterruptedly until offset more frequently perhaps the paroxysm is interrupted at ... intervals by several sinus beats or by a short

...

of ectopic beats separated from one another by one or two sinus beats (Figs 292 and 293) The first ectopic beat of each brief paroxysm appears prematurely after the preceding sinus beat and is coupled to it by ... The last beat of every

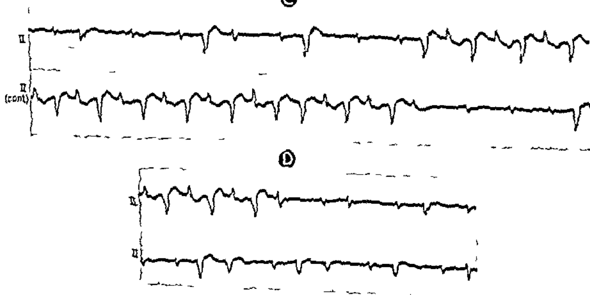
...

Paroxysmal ventricular tachycardias ... somewhat in their electrocardiographic features The variations most commonly observed are those described below

1 ... the appearance of the ventricular ... beat ... 130– ...

2 On the other hand some ventricular ta... cardias are characterized by an irregular rhythm and by changes in the appearance of the ventricular deflections from beat to beat

3 Sometimes the ectopic ventricular beats are so widened and distorted that the individual deflection of the QRS complex are difficult to distinguish from the T wave (Fig 295) Moreover the appearance of the ectopic beats and the intervals separating them often change from beat to beat As the ventricular tachycardia approaches the fibrillatory stage the ectopic beats gradually take the form of regular undu



of His

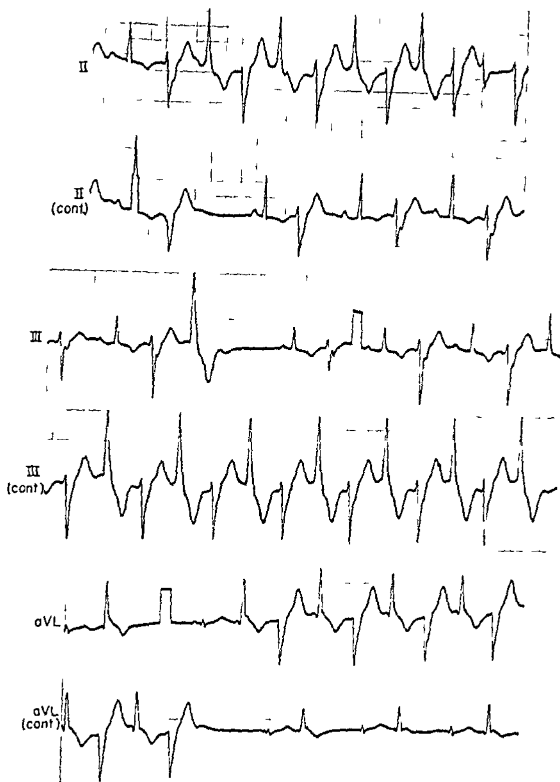
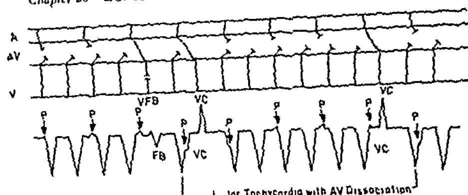


Fig 297 —Alternating bidirectional tachycardia This example is somewhat unusual in that sinus rhythm persists during the tachycardia as the result of atrioventricular dissociation In addition it can be seen in the two continuous strips of le
widening (
III the co
the continuous strip of lead III below they p
tachycardia Thus it seems that the upright R v
originate above the bifurcation of the bundle c
flections is not clear although it may be in the ventricles In summary this and the preceding three examples of alternating bidirectional tachycardia suggest that this type of tachycardia actually represents a heterogeneous group of cardiac rhythms which may differ in mechanism and their site of origin



lar Tachycardia with AV Dissociation

not, it is impossible to di
be made on the basis of t
normal duration (
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Ventricular aberr

be assumed that the bizarre appearance of the
gin of the ectopic beats rather than defective intraventricular conduction of atrioventricular nodal impulses

lating waves characteristic of prefibrillatory tachycardia or ventricular flutter

4 On rare occasions particularly in severe digitalis intoxication a form of ventricular tachycardia is encountered which is unique in that alternate ventricular complexes are oppositely directed It is called *bidirectional ventricular tachycardia* (or better *alternating bidirectional tachycardia*) and when present is considered a poor prognostic sign Carotid sinus stimulation has been noted at times to abolish all similarly directed deflections without affecting those oppositely directed The mechanism underlying bidirectional ventricular tachycardia is not known, but the explanation currently favored is that the ectopic impulses arise above the bifurcation of the bundle and are conducted alternately down the left and right bundle branches If this theory is correct, then a bidirectional tachycardia may have its origin in either the atrioventricular node or the ventricles If the tachycardia has a ventricular origin the ectopic focus is located in the common bundle and probably not below it (Figs 294 296 and 297)

Retrograde activation of the atria by ectopic ventricular impulses is an unusual event in ventricular tachycardia, although the frequency with which it occurs is perhaps underestimated The fact that retrograde activation of the atria during ventricular tachycardia is not always recognized is not surprising in view of the difficulty experienced in ventricular tachycardias in identifying sinus P waves much less retrograde P waves When it is possible to recognize P waves they are usually found to be sinus P waves which appear at a rate and rhythm independent of the rate and rhythm present in the ventricles While sinus bradycardia or severe sinus inhibition or arrest may accompany ventricular tachycardia in the dying heart, the atrial rhythm in most instances is a relatively fast sinus rhythm or sinus tachycardia The cause of the enhanced rhythmicity of the sinus pacemaker is not definitely established but changes in the nutrition and oxygenation of the sinoatrial node have been implicated by some investigators In any event the result is that the sinus and ectopic impulses interfere with each other in or near the atrioventricular junctional tissues and complete or incomplete atrioventricular dissociation is the outcome On rare occasions in incomplete dissociation a sinus impulse may be able to capture the ventricles and interrupt the tachycardia for a single beat The ventricular capture beat which appears may provide one of the few reliable clues to the correct identification of a paroxysmal tachycardia characterized by abnormal wide QRS complexes (Fig 298) In such a tachycardia the two

diagnostic alternatives to be differentiated are ventricular tachycardia and atrioventricular nodal tachycardia with ventricular aberration The following electrocardiographic findings favor in varying degree the diagnosis of ventricular tachycardia (Fig 298)

Complete ventricular capture without aberration of the conducted beat —When a sinus impulse captures the ventricles completely the ectopic impulse discharged shortly thereafter does not participate at all in ventricular excitation For this to happen it is evident that the sinus impulse must of necessity initiate ventricular excitation prematurely—that is prior to discharge of the ectopic impulse Thus the premature onset of the ventricular capture beat allows the ventricles a shorter recovery period than usual If despite this fact the ventricular capture beat fails to show ventricular aberration it is hardly likely that the abnormal duration and appearance of the ectopic ventricular beats elsewhere in the same lead strip are the result of aberrant intraventricular conduction of nodal impulses Therefore a ventricular capture beat with a QRS interval of 0 10 second or less strongly favors the diagnosis of ventricular tachycardia

Incomplete ventricular capture with the appearance of a ventricular fusion beat —If ventricular capture by the sinus beat is incomplete excitation processes initiated by the sinus and ectopic ventricular impulses proceed simultaneously in different directions through the myocardium Since the ventricular fusion beat which results can be produced only by fusion of a supraventricular impulse with an impulse originating in the ventricles (the principal exception to this rule is ventricular pre excitation occurring in the Wolff Parkinson White syndrome) the recognition of one or more ventricular fusion beats during a paroxysmal tachycardia establishes as a general rule its identity as a ventricular tachycardia and excludes the possibility of atrioventricular nodal tachycardia

Ventricular extrasystoles in antecedent or subsequent electrocardiograms —If single ventricular extrasystoles are observed in an electrocardiogram recorded shortly before onset or after offset of a paroxysmal tachycardia, the probability is that the tachycardia is of ventricular origin

Relatively slow rate and slight irregularity of the tachycardia —In general the slower and the more irregular the ventricular rhythm during the tachycardia the more likely it is that ventricular tachycardia is *present and the less likely is the diagnosis of atrioventricular nodal tachycardia with ventricular aberration*

Clinical Significance of Ventricular Tachycardia

Ventricular tachycardia is rarely observed in the absence of cardiac disease It may appear as a complication of recent myocardial infarction severe hypertensive or arteriosclerotic heart disease congestive heart failure and digitalis therapy if intoxication occurs Paroxysmal ventricular tachycardia requires prompt therapy for several reasons (*a*) Since ventricular tachycardia occurs almost invariably in a severely damaged heart the rapid ventricular rate is all the more likely to compromise cardiac function and lead to the onset of acute cardiac decompensation and pulmonary edema or to Stokes Adams attacks (consisting of shock and syncope sometimes associated with convulsions) (*b*) An even greater hazard posed by ventricular tachycardia is that it is sometimes the precursor of ventricular fibrillation which is with few exceptions a fatal arrhythmia (*c*) A less frequent cause of Stokes Adams attacks is cardiac standstill following abrupt offset of ventricular tachycardia

ATRIAL FLUTTER AND ATRIAL FIBRILLATION

Ectopic atrial rhythms with rates exceeding the range of rates usually encountered in paroxysmal supraventricular tachycardia are of two closely related types atrial flutter and atrial fibrillation These two rhythms differ mainly in the following respects (*a*) In atrial flutter the configuration of the atrial flutter (F) waves and the F–F intervals remain constant from beat to beat while fibrillation is characterized by undulating atrial (f) waves which vary widely in contour amplitude and timing (*b*) Atrial flutter waves appear at rates of 250–350 per minute although slower rates are frequently observed during quinidine administration In atrial fibrillation the rate of the fibrillatory oscillations (f) is usually impossible to measure accurately but is certainly faster than 350 beats per minute

The close interrelationship of atrial flutter and fibrillation has a firm foundation in clinical experience and experimental observations Clinically the two rhythms are often observed on different occasions in

the same individual digitalis commonly converts atrial flutter into fibrillation and finally atrial fibrillation frequently passes through a period of flutter shortly before conversion to sinus rhythm by quinidine. *Experimental evidence for such an affinity between atrial flutter and fibrillation is fairly extensive and will be summarized later in discussing the mechanism of flutter and fibrillation, on pages 442 and 443*

The manner in which atrial flutter and fibrillation arise is one of the most controversial and complicated aspects of cardiac pathophysiology From a strictly practical standpoint, it is not of great importance to the clinical electrocardiographer whether the specific mechanism involved is circus movement (pp 442–443) rapid focal or multifocal impulse formation or multiple re-entries of one or many impulses In fact, none of these hypothetical mechanisms have as yet been irrefutably proved For these reasons consideration of the theories cited above will be limited to a brief recapitulation at the end of the chapter but pertinent references listed in the bibliography may be consulted for more detailed presentations In the following discussion it will, for simplicity and convenience be assumed that atrial flutter and fibrillation originate in a single ectopic atrial focus where impulses are formed and discharged much faster than in paroxysmal supraventricular tachycardia

Because of their short refractory period, atrial muscle fibers are able to respond regularly to ectoptic atrial impulses discharged at rates approaching 350 beats per minute and can conduct the impulses just as rapidly as normally Since the atria are therefore activated in an identical manner by each excitation impulse the atrial deflections which are produced retain the same configuration and cycle length from beat to beat. Impulse formation in an ectopic atrial focus at a rate of 250 beats per minute or faster and an unchanging pattern of intra atrial conduction of each ectopic impulse are the characteristics distinguishing atrial flutter from paroxysmal atrial tachycardia on the one hand, and atrial fibrillation on the other

If an ectopic atrial focus fires off too rapidly and/or the refractory period of the atrial myocardium is abnormally prolonged the fibers may not be able to recover completely from excitation by one impulse before the next is discharged In this event the refractory atrial muscle responds irregularly or not at all, to excitatory stimuli Successive ectopic impulses may be blocked in some areas and not in others or they may spread slowly erratically and incompletely through the atrial myocardium The end result is that the atria remain in a state of continuous chaotic electrical activity which is reflected in the electrocardiogram by constant wavelike undulations of the base line These fibrillatory waves vary markedly in contour and size and appear irregularly at a rate exceeding 350 beats per minute Atrial fibrillation therefore represents failure of intra atrial conduction in the

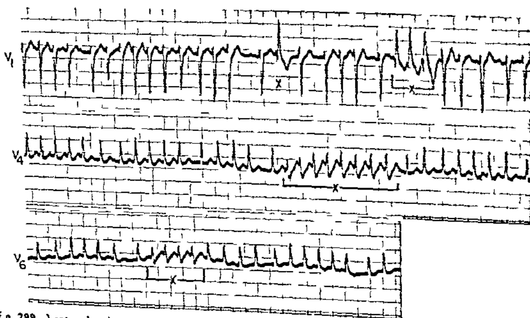


Fig 299 —Ventricular aberration of conducted beats in atrial fibrillation simulating paroxysms of ventricular tachycardia. Frequently when there is aberrant intraventricular conduction the ventricular beats (X) affected have a configuration resembling that observed in right bundle branch block.

presence of a very rapid ectopic atrial rhythm Depressed atrial conductivity secondary to digitalis myocardial disease or other factors is frequently responsible for atrial flutter changing to fibrillation at less rapid rates of stimulation than would otherwise be the case

Aberrant intraventricular conduction —Just as may happen to any impulse arriving in the ventricles shortly after a previous impulse flutter or fibrillatory impulses may sometimes be conducted to the ventricles in such close succession that recovery is not complete and excitation therefore spreads in an aberrant fashion through the myocardium This leads to aberration of the ventricular deflection produced by the conducted supraventricular impulse so that the QRS complex differs from others in duration and/or configuration Aberrant intraventricular conduction of flutter or fibrillatory impulses is the consequence of incomplete recovery of ventricular muscle and conducting pathways and usually involves beats which terminate a short cycle particularly if the short cycle follows a relatively long one (see discussion of the effect of cycle length on the refractory period on p 354) Sometimes a long pause in the ventricular rhythm is terminated by a ventricular beat which may or may not show minimal to moderate aberration This can usually be proved to be an escape beat by virtue of the fact that whenever a beat of this type appears in the record it follows the last conducted beat by the same constant interval Ordinarily the escape beat originates in the atrioventricular node

It is frequently difficult to differentiate conducted beats with ventricular aberration from ectopic ventricular beats especially in atrial fibrillation This becomes particularly important when a rapid run of abnormal ventricular deflections is observed during atrial fibrillation (Fig 299) The therapeutic approach is quite different if these beats are conducted supraventricular beats with ventricular aberration as opposed to a short paroxysm of ventricular tachycardia In attempting to differentiate the two types of abnormal ventricular beats the following generalizations are sometimes helpful (*a*) Ventricular aberration tends to produce a QRS configuration resembling that of right bundle branch block (*b*) Conducted beats with aberrant intraventricular conduction are likely to vary in appearance as the degree of aberration fluctuates while the configuration of ventricular extrasystoles originating in a single focus usually remains constant (*c*) Unifocal ventricular extrasystoles typically are coupled to the preceding beat by a fixed interval but the cycles preceding conducted beats with aberration differ in length (*d*) Ectopic ventricular beats in flutter and fibrillation are often followed by a pause produced by retrograde penetration of the atrioventricular junctional tissues by the ectopic impulse

Atrial Flutter

While clinically the rate and regularity or irregularity of the ventricular rhythm are justifiably stressed in the auscultatory diagnosis of atrial flutter and fibrillation the electrocardiographic diagnosis of these two rhythms does not rest on these features The contour rate and regularity of the atrial deflections provide the basis for the diagnosis of atrial flutter or fibrillation regardless of the characteristics of the ventricular rhythm

The flutter (F) waves appear at rates of 250–350 per minute but rates below 200 waves per minute may be observed during the course of quinidine therapy (Fig 300) The F waves can be recognized by their uniform configuration amplitude and timing in a given lead Typically every flutter wave consists of two oppositely directed components corresponding to the P wave of atrial excitation and the following Ta wave of atrial repolarization The prominence of the Ta waves in particular seems directly related to the atrial rate and increases as the rate accelerates Thus the sawtooth or picket fence oscillations of the base line characterizing flutter which is produced by relatively large alternating upward and downward components of the F waves are a reflection of the rate of atrial excitation and not the mechanism by which this occurs Quinidine tends to widen the saw tooth F waves and slows their rate until frequently they come to present more of a sine wave appearance

In some leads and occasionally in all leads the flutter waves may be recorded as uniphasic deflections separated by isoelectric intervals and may be flat upright or inverted Monophasic F waves are observed in slow atrial flutter chiefly Leads II III aVF V_1 and V_2 ordinarily register the most prominent F waves However it is not unusual for only one or two of these leads to show identifiable F waves the atrial mechanism being poorly defined in all others

Since the atrioventricular junctional tissues normally have a longer refractory period than either the atrial or ventricular myocardium atrioventricular conductivity becomes the limiting factor governing the ventricular response in flutter As a general rule the atrioventricular conducting pathway cannot respond to more than 200–250 impulses per minute Impulses

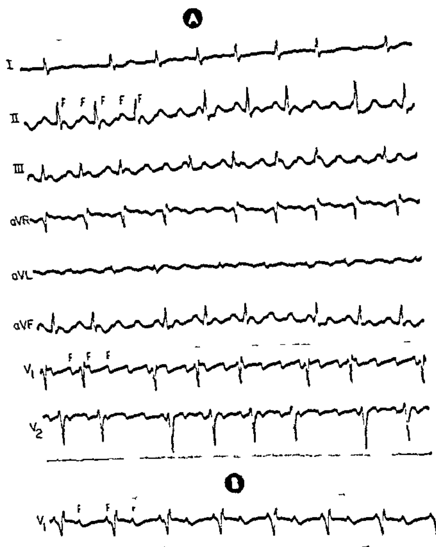


Fig 300 —Effect of quinidinization on atrial flutter Electrocardiogram **A** was recorded after the patient had been fully digitalized and early in the course of quinidinization Before starting quinidine therapy the electrocardiogram showed atrial flutter waves (*F*) with an atrial rate of 300 beats per minute while the above electrocardiogram shows an atrial rate of about 200 beats per minute illustrating the slowing effect of quinidine on the rate of atrial flutter Note that there is varying atrioventricular response in **A** presumably reflecting the depressant effect of digitalis on atrioventricular conduction The strip of lead V_1 in **B** was recorded 24 hours later when peak levels of quinidine had been attained As a result of the marked quinidine effect the atrial rate has slowed to 140 beats per minute but the basic configuration of the flutter waves in lead V_1 has not changed Note also that there is now 2:1 atrioventricular response The decreased ratio of atrioventricular response in this instance is probably related to two factors (1) the general slowing of the rate of the atrial flutter and (2) the vagolytic effect of quinidine which tends to abolish the indirect effect of digitalis on atrioventricular conduction

presence of a very rapid ectopic atrial rhythm De pressed atrial conductivity secondary to digitalis myocardial disease or other factors is frequently responsible for atrial flutter changing to fibrillation at less rapid rates of stimulation than would otherwise be the case

Aberrant intraventricular conduction —Just as may happen to any impulse arriving in the ventricles shortly after a previous impulse flutter or fibrillatory impulses may sometimes be conducted to the ventricles in such close succession that recovery is not complete and excitation therefore spreads in an aberrant fashion through the myocardium This leads to aberration of the ventricular deflection produced by the conducted supraventricular impulse so that the QRS complex differs from others in duration and/or configuration Aberrant intraventricular conduction of flutter or fibrillatory impulses is the consequence of incomplete recovery of ventricular muscle and conducting pathways and usually involves beats which terminate a short cycle particularly if the short cycle follows a relatively long one (see discussion of the effect of cycle length on the refractory period on p 354) Sometimes a long pause in the ventricular rhythm is terminated by a ventricular beat which may or may not show minimal to moderate aberration This can usually be proved to be an escape beat by virtue of the fact that whenever a beat of this type appears in the record it follows the last conducted beat by the same constant interval Ordinarily the escape beat originates in the atrioventricular node

It is frequently difficult to differentiate conducted beats with ventricular aberration from ectopic ventricular beats especially in atrial fibrillation This becomes particularly important when a rapid run of abnormal ventricular deflections is observed during atrial fibrillation (Fig 299) The therapeutic approach is quite different if these beats are conducted supraventricular beats with ventricular aberration as opposed to a short paroxysm of ventricular tachycardia In attempting to differentiate the two types of abnormal ventricular beats the following generalizations are sometimes helpful (*a*) Ventricular aberration tends to produce a QRS configuration resembling that of right bundle branch block (*b*) Conducted beats with aberrant intraventricular conduction are likely to vary in appearance as the degree of aberration fluctuates while the configuration of ventricular extrasystoles originating in a single focus usually remains constant (*c*) Unifocal ventricular extrasystoles typically are coupled to the preceding beat by a fixed interval but the cycles preceding conducted beats with aberration differ in length (*d*) Ectopic ventricular beats in flutter and fibrillation are often followed by a pause produced by retrograde penetration of the atrioventricular junctional tissues by the ectopic impulse

Atrial Flutter

While clinically the rate and regularity or irregularity of the ventricular rhythm are justifiably stressed in the auscultatory diagnosis of atrial flutter and fibrillation the electrocardiographic diagnosis of these two rhythms does not rest on these features The contour rate and regularity of the atrial deflections provide the basis for the diagnosis of atrial flutter or fibrillation regardless of the characteristics of the ventricular rhythm

The flutter (F) waves appear at rates of 250–350 per minute but rates below 200 waves per minute may be observed during the course of quinidine therapy (Fig 300) The F waves can be recognized by their uniform configuration amplitude and timing in a given lead Typically every flutter wave consists of two oppositely directed components corresponding to the P wave of atrial excitation and the following Ta wave of atrial repolarization The prominence of the Ta waves in particular seems directly related to the atrial rate and increases as the rate accelerates Thus the saw tooth or picket fence oscillations of the base line characterizing flutter which is produced by relatively large alternating upward and downward components of the F waves are a reflection of the rate of atrial excitation and not the mechanism by which this occurs Quinidine tends to widen the saw tooth F waves and slows their rate until frequently they come to present more of a sine wave appearance

In some leads and occasionally in all leads the flutter waves may be recorded as uniphasic deflections separated by isoelectric intervals and may be flat up right or inverted Monophasic F waves are observed in slow atrial flutter chiefly Leads II III aVF V_1 and V_2 ordinarily register the most prominent F waves However it is not unusual for only one or two of these leads to show identifiable F waves the atrial mechanism being poorly defined in all others

Since the atrioventricular junctional tissues normally have a longer refractory period than either the atrial or ventricular myocardium atrioventricular conductivity becomes the limiting factor governing the ventricular response in flutter As a general rule the atrioventricular conducting pathways cannot respond to more than 200–250 impulses per minute Impulses

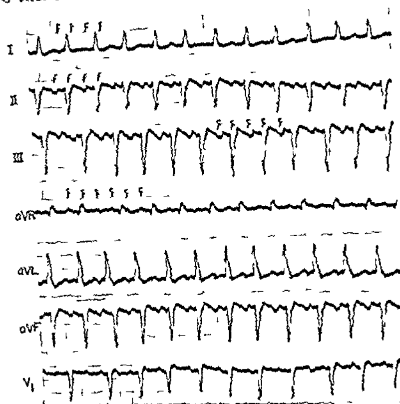


Fig 302 – Atrial flutter with 2 1 atrioventricular response The flutter waves (*F*) appear at a rate of about 260 beats per minute The saw tooth appearance of the flutter waves is best demonstrated in leads II III and aVF Since the flutter waves are almost entirely upright in lead aVR it is highly likely that in this instance the atrial flutter is originating low in the atria (In this text, the single ectopic focus theory of the genesis of atrial flutter is followed) Note that, in each lead strip atrioventricular conduction *occasionally* changes from 2 1 to 3 1

arriving at the atrioventricular node at a more rapid rate may find it in its normal refractory period and may not be conducted. Atrioventricular interference is the mechanism responsible for 2 1 atrioventricular response the ratio so commonly observed in untreated atrial flutter (Fig 301 A) This is an example of *physiologic blocking* in that every second impulse arrives at the atrioventricular node before it has recovered from excitation by the preceding beat The atrioventricular junctional tissues are just as responsive and recover just as rapidly as normally but the atrial impulses follow each other in too close succession Digitalis therap[illegible]

atrioventricular conductivity and thereby superimpose a pathologic block on the pre existing physiologic block As a result the 2 1 ratio of atrioventricular response is further modified at the level of the block and a 4 1 or 6 1 ratio may appear a 3 1 atrioventricular ratio being relatively uncommon (Fig 301 A, *B and C*) With a fixed ratio of atrioventricular response the F–R intervals (the interval between corresponding points on the flutter waves and onset of the following ventricular complexes) remain of constant length If the relationship between the flutter waves and the ventricular beats varies but the R–R intervals remain constant, complete atrioventricular block is likely to be present

Irregular atrioventricular response is far more common in digitalis treated atrial flutter than is fixed atrioventricular response the atrioventricular ratios in a given lead strip for example fluctuating erratically between the extremes of 2 1 and 8 1 Variations in *atrioventricular conductivity are evidenced not only* by changing ratios of atrioventricular response but also by the inconstant F–R intervals of the conducted beats Generally the interval between a conducted ventricular beat and the preceding F wave (either its apex or nadir) is *longer following short cycles* and shorter following long cycles A Wenckebach type of progressive prolongation of the F–R intervals may be observed in successive cycles having the same conduction ratio (Fig 301 C) With the occurrence of a longer R–R cycle the F–R *interval shortens and* the sequence commences again From the preceding facts it becomes evident that the ventricular rhythm in atrial flutter may sometimes be just as irregular as in fibrillation and that this finding does not differentiate the two atrial rhythms (*Figs 302–304*)

To explain the irregularities in atrioventricular response it has been postulated that some flutter

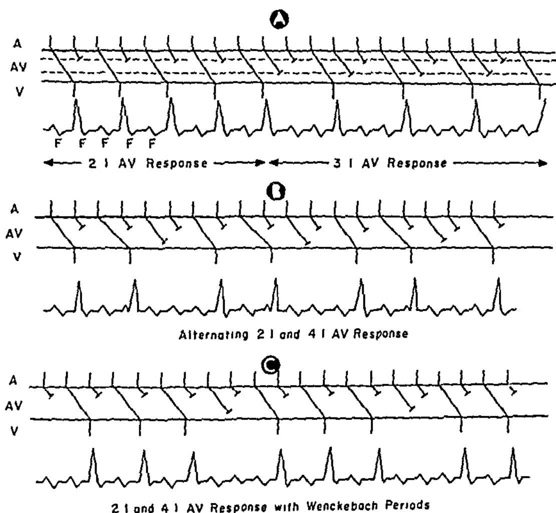


Fig 301 —Mechanisms of atrioventricular response in atrial flutter In the first half of diagram A atrial flutter with 2 1 atrioventricular response is depicted Because of the rapid atrial rate every second flutter wave (*F*) arrives at the atrioventricular node in the refractory period of the previously conducted atrial beat and is therefore not conducted It is commonly thought that the nonconducted atrial impulse is blocked at a relatively high level in the atrioventricular junctional tissues In reality 2 1 atrioventricular response in atrial flutter is not necessarily indicative of atrioventricular

the mechanism of alternating 2 1 and 4 1 atrioventricular response in atrial flutter is illustrated This mechanism frequently leads to a pseudobigeminal rhythm in the ventricle Note that the atrioventricular conduction time of the second

beats can be seen to lengthen gradually until finally 4 1 atrioventricular response appears

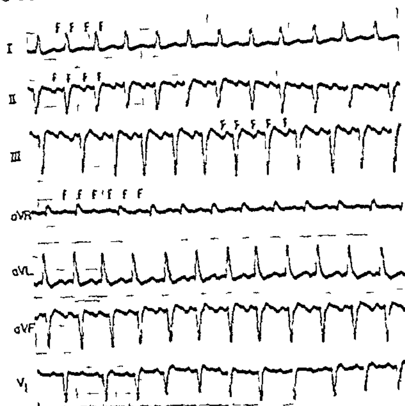


Fig 302.—Atrial flutter with 2:1 atrioventricular response The flutter waves (*F*) appear at a rate of about 60 beats per minute The saw tooth appearance of the flutter waves is best demonstrated in leads II III and aVF Since the flutter waves are almost entirely upright in lead aVR it is highly likely that in this instance the atrial flutter is originating low in the atria (In this text the single ectopic focus theory of the genesis of atrial flutter is followed.) Note that, in each lead strip atrioventricular conduction occasionally changes from 2:1 to 3:1

arriving at the atrioventricular node at a more rapid rate may find it in its normal refractory period and may *not* be conducted Atrioventricular interference is the mechanism responsible for 2:1 atrioventricular response the ratio so commonly observed *in untreated* atrial flutter (Fig 301 A) This is an example of *physiologic blocking*, in that every second impulse arrives at the atrioventricular node before it has recovered from excitation by the preceding beat The atrioventricular junctional tissues are just as responsive and recover just as rapidly as normally but the atrial impulses follow each other in too close succession. Digitalis therapy carotid sinus stimulation inflammatory or degenerative changes in the atrioventricular node and other such influences may depress atrioventricular conductivity and thereby superimpose a pathologic block on the pre existing physiologic block As a result the 2:1 ratio of atrioventricular response is further modified at the level of the block and a 4:1 or 6:1 ratio may appear a 3:1 atrioventricular ratio being relatively uncommon (Fig 301 A B and C) With a fixed ratio of atrioventricular response the F–R intervals (the interval between corresponding points on the flutter waves and onset of the following ventricular complexes) remain of constant length If the relationship between the flutter waves and the ventricular beats varies but the R–R intervals remain constant complete atrioventricular block is likely to be present

Irregular atrioventricular response is far more common in digitalis treated atrial flutter than is fixed atrioventricular response the atrioventricular ratios in a given lead strip for example fluctuating erratically between the extremes of 2:1 and 8:1 Variations in atrioventricular conductivity are evidenced not only by changing ratios of atrioventricular response but also by the inconstant F–R intervals of the conducted beats Generally the interval between a conducted ventricular beat and the preceding F wave (either its apex or nadir) is longer following short cycles and shorter following long cycles A Wenckebach type of progressive prolongation of the F–R intervals may be observed in successive cycles having the same conduction ratio (Fig 301 C) With the occurrence of a longer R–R cycle the F–R interval shortens and the sequence commences again From the preceding facts it becomes evident that the ventricular rhythm in atrial flutter may sometimes be just as irregular as in fibrillation and that this finding does not differentiate the two atrial rhythms (Figs 302–304)

To explain the irregularities in atrioventricular response it has been postulated that some flutter

impulses although failing to emerge from the atrioventricular node into the ventricles penetrate the atrioventricular junctional tissues more deeply than other block impulses Thus nonconducted flutter impulses may produce negligible or marked refractoriness of the atrioventricular junctional tissues depending on whether they are blocked high in the atrioventricular node or penetrate more deeply before being blocked If the latter event occurs the prolonged refractory state may result in slower conduction of the next impulse or in failure of the following one or more flutter beats to be conducted This phenomenon is called *concealed atrioventricular conduction* and is thought to be responsible for the variations in the atrioventricular response ratios and F–R intervals and for the appearance of 3 1 and 5 1 atrioventricular conduction in treated atrial flutter The mechanism underlying this phenomenon is atrioventricular interference

Impure Atrial Flutter or Atrial Flutter Fibrillation

No definite boundary demarcates atrial flutter from fibrillation Between the two there exists only a vague transitional stage in which the atrial rhythm exhibits features of both flutter and fibrillation Impure atrial flutter or *flutter fibrillation* the transitional rhythm linking typical flutter and typical fibrillation is characterized by slight variations in the appearance and regularity of the F waves and an atrial rate approaching that of fibrillation For all intents and purposes impure atrial flutter and coarse atrial fibrillation are so closely related that there seems little practical value in distinguishing one from the other

Atrial Fibrillation

In fibrillation the atrial (f) waves are irregular in rhythm dissimilar in appearance and of lower ampli

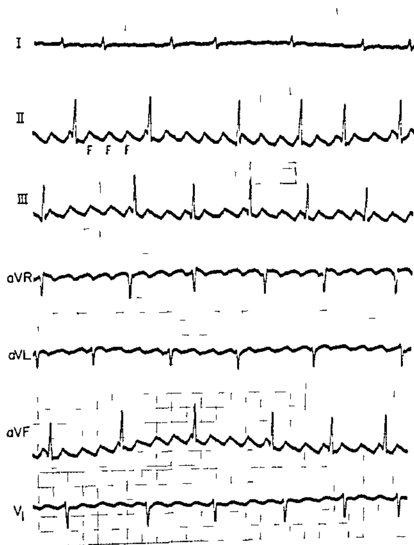


Fig 303 –Atrial flutter with ratios of atrioventricular response varying from 2 1 to 5 1 The rate of atrial flutter waves (*F*) is about 250 beats per minute

tude ordinarily than flutter waves The fibrillatory waves occur at rates varying between 400 and 700 per minute and may be prominent in some leads and not in others As a general rule very rapid fibrillatory rates are associated with low amplitude f waves while coarse f waves tend to accompany slower rates Sometimes there is so little disturbance of the base line that the fibrillatory waves cannot be differentiated with any degree of certainty from oscillations due to extraneous artefact In such an instance a presumptive diagnosis of atrial fibrillation can be made if P waves are not demonstrable and the ventricular rhythm is irregular

...ilts from ...pulses at ...ariations

in atrioventricular conduction of the impulses Some of the atrial impulses are conducted to the ventricles and others are blocked at different levels in the atrioventricular junction tissues The variable depth of penetration of the blocked impulses affects the manner in which subsequent impulses are conducted Thus concealed atrioventricular conduction is another factor contributing to the irregular ventricular rhythm in atrial fibrillation (Fig 305)

The atrioventricular pathway is normally able to conduct only 200–250 impulses per minute Inasmuch as the fibrillatory impulses arrive at the node at a far more rapid rate atrioventricular interference is always present in atrial fibrillation and may be the major mechanism regulating ventricular response when a rapid ventricular rate is encountered Both interference and atrioventricular block whether due to digitalis therapy or to pathologic changes in the nodal tissues must be implicated when the ventricular rate in atrial fibrillation is within the range of 60–100 beats per minute (Fig 306) Slower ventricular rhythms are usually indicative of higher degrees of atrioventricular block and if perfectly regular of complete atrioventricular block

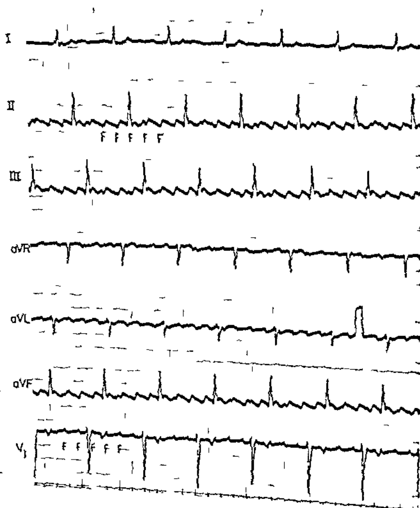


Fig 304 —Atrial flutter with a rate of about 275 beats per minute and 4:1 atrioventricular response the flutter waves being labeled F

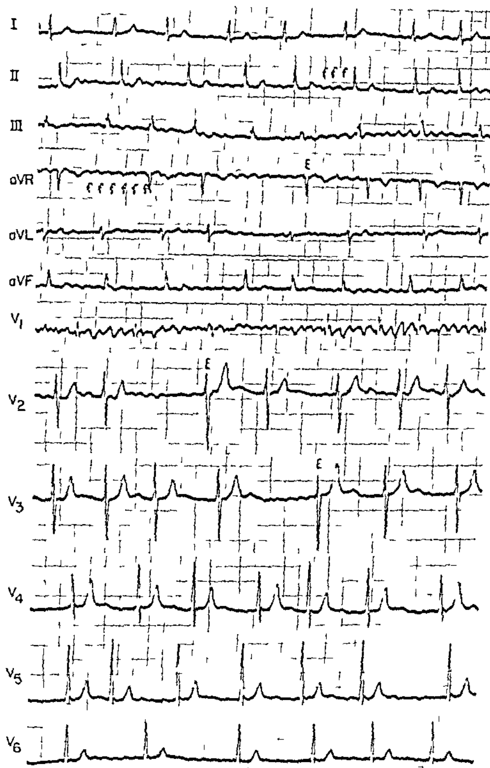
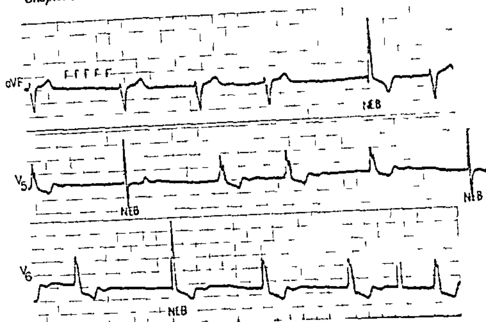


Fig 305—Atrial fibrillation. The basis for the diagnosis of atrial arrhythmia in this instance consists solely of the finding of rapid atrial oscillations (*f*) which are irregular in both appearance and rate of occurrence. The irregular ventricular response in atrial fibrillation can probably be ascribed to varying degrees of penetration of the atrioventricular nodal tissues by the fibrillatory impulses and the consequent variation in the rates of atrioventricular conduction of the atrial impulses producing ventricular beats. The ventricular complexes (*E*) in leads aVR, V and V are preceded by an interval of the same length in each instance. These ventricular beats must therefore be atrioventricular nodal escape beats. Note also that the nodal escape interval is the longest R–R interval in the electrocardiogram. Parenthetically it might be added that this electrocardiogram displays evidence of right ventricular enlargement (RSR deflections in lead V_1) and was recorded from a patient known to have mitral stenosis.



F 306 Atrial fibrillation in which the conducted ventricular beats present the appearance of left bundle branch … a different appearance and a relatively normal …

beats The symbol f indicates the fibrillatory waves

Effect of Vagal Stimulation in Flutter and Fibrillation

Carotid sinus stimulation the Valsalva maneuver or other measures increasing vagal tone may produce the following changes during atrial flutter and fibrillation

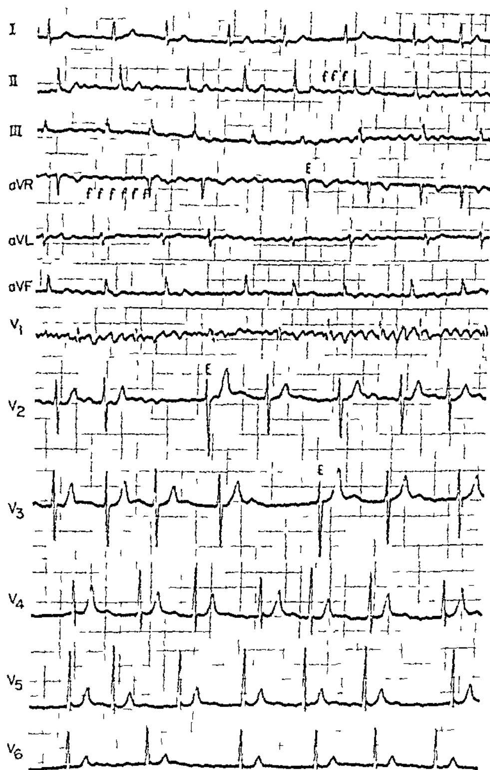
In atrial flutter – (a) The atrial rate may increase or rarely the flutter may be converted to fibrillation (b) The main effect of vagal stimulation is to depress atrioventricular conductivity and by so doing to slow the rate of ventricular response In atrial flutter the ventricular rate falls abruptly and irregularly as the ratio of atrioventricular response shifts—for example from 2 1 to 6 1 to 4 1 to 8 1 etc This sequence is reversed so that there is an irregular return to the former rapid ventricular rate Atrioventricular node depression persists only as long as carotid sinus stimulation is continued although sometimes atrioventricular conductivity is restored before termination of the procedure

In atrial fibrillation —As in flutter vagal stimulation depresses atrioventricular conduction and decreases the number of atrial impulses transmitted to the ventricles Although there is irregular slowing of the ventricular rhythm the abrupt jumps in ventricular rate observed in flutter as the atrioventricular rate is halved quartered etc are not characteristic of atrial fibrillation

Measures increasing vagal tone—carotid sinus stimulation in particular—are important aids to the recognition of atrial flutter and fibrillation In flutter with 2 1 atrioventricular response every other flutter wave may be hidden in a ventricular deflection so that the rhythm may be interpreted as paroxysmal supraventricular tachycardia By slowing the ventricular rate carotid sinus stimulation usually permits flutter waves obscured by QRS or T waves to be visualized This holds equally for atrial fibrillation with fast ventricular rates *When the ventricular rhythm is very rapid in* fibrillation, the R–R intervals sometimes vary little if at all in length In the absence of an irregular ventricular rhythm and identifiable atrial deflections the diagnosis of atrial fibrillation would be difficult to establish without resort to carotid sinus stimulation This slows the ventricular rhythm and more clearly demonstrates the atrial mechanism just as in flutter in addition the irregularity of the ventricular rhythm is accentuated (Figs 307 and 308)

Clinical Aspects of Atrial Flutter and Fibrillation

Both atrial flutter and fibrillation may occur in a paroxysmal transient form or in an established chronic

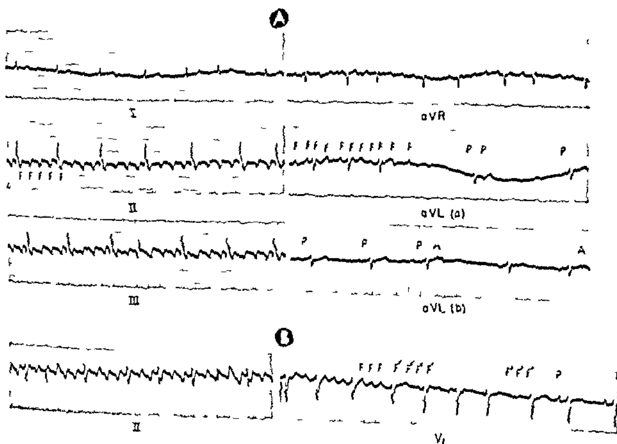


... ar enlargement (RSR deflections in
... t ent known to have mitral stenosis

form Atrial fibrillation is one of the most commonly noted cardiac arrhythmias and is said to occur in about 70% of cases of severe heart failure The incidence of atrial flutter is much lower the ratio of atrial flutter to fibrillation being in the order of 1 10

Both arrhythmias ordinarily are associated with cardiac disease—*atrial flutter almost invariably so However many investigators have recorded cases of* atrial fibrillation usually of the paroxysmal type occurring *in* the apparent absence of cardiac disease. In several series such cases comprised approximately 6% of the total number of patients with atrial fibrillation In general atrial flutter and fibrillation tend to be superimposed on the same types of heart disease (coronary arteriosclerosis hypertension and rheumatic mitral valvular disease)

Prolongation of the P-R interval produced by these and other types of cardiac disease has been said to predispose to subsequent onset of atrial fibrillation *It is not surprising therefore* that after reversion of atrial fibrillation *to sinus rhythm the P-R interval frequently remains prolonged independently of any* drug effect Both arrhythmias may complicate myocardial infarction but neither is ordinarily seen in syphilitic heart disease in the absence of coexisting diseases like those listed above Thyrotoxicosis may precipitate either arrhythmia but atrial fibrillation is the more commonly noted of the two Atrial arrhyt



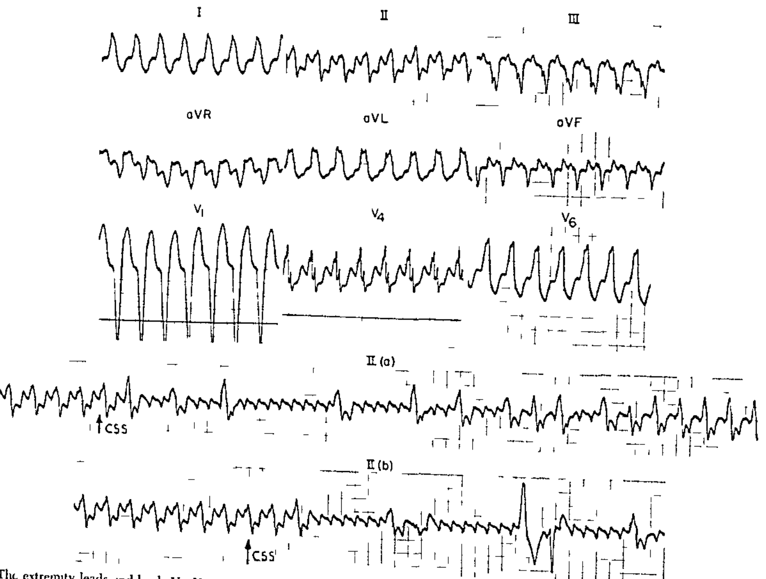


Fig 307 —The extremity leads and leads V1, V4 and V6 in the upper half of the figure show a rapid ventricular rhythm, the rate being about 170 beats per minute. The ventricular complexes are deformed in a manner suggesting left bundle branch block and there appear to be atrial deflections preceding each ventricular complex. Therefore, at this point, the electrocardiogram might be interpreted as showing paroxysmal atrial tachycardia with left bundle branch block, or if the identification of the deflections preceding the ventricular complexes as atrial beats is not accepted, the rhythm would have to be interpreted as paroxysmal ventricular tachycardia. The two long strips of lead II shown at the bottom were recorded a short time later. In lead II (*a*) the first portion of the lead shows a tachycardia identical with that in lead II above. However, with carotid sinus stimulation (CSS at arrow) the resulting slowing of the ventricular rate due to decreased atrioventricular conduction reveals rapid atrial flutter waves. Note the irregular escape of atrioventricular conduction from the effects of carotid sinus stimulation later in the same lead strip. Lead II (*b*) demonstrates the same response to carotid sinus stimulation as was described in lead II (*a*). This electrocardiogram illustrates the value of carotid sinus stimulation in elucidating the mechanism of a paroxysmal tachycardia.

pathway to initiate another cycle As long as the activation front is preceded by *nonrefractory* muscle the circus movement continues without interruption In short, the circus movement mechanism requires that there be a gap of nonrefractory responsive muscle separating the head of the mother wave from its tail *The success of quinidine* in terminating atrial flutter and fibrillation has been attributed to the fact that it prolongs the refractory period of atrial muscle (more precisely to its slowing of conduction recovery) which, in effect, abolishes the indispensable "gap" of recovered myocardium preceding the mother wave The circus movement subsides as soon as the mother wave encounters refractory myocardium

In atrial flutter the path traversed by the mother circus is thought to be longer than in fibrillation and thus the atrial oscillations are slower in flutter than in fibrillation It is also believed that the atria recover more rapidly in flutter than in fibrillation, so that the mother and daughter waves tend to follow a relatively constant pathway from cycle to cycle This circumstance produces flutter waves of identical shape and timing in a given lead On the other hand the atrial recovery process is slower in atrial fibrillation Consequently the pathways traversed by the mother and daughter circus waves vary from cycle to cycle, and this is reflected in the variable appearance of the fibrillatory oscillations

Multiple re-entry —The mechanism of re-entry has already been described with reference to coupled ectopic beats occurring singly and to paroxysmal ectopic tachycardia In flutter and fibrillation according to Katz and Pick the initiating sinus or ectopic impulse

by virtue of numerous interferences in the heart and reentries from a number of points multiplies the number of daughter impulses to varying degrees Each impulse continually wanders about the syncytium wherever it finds responsive muscle not yet stimulated by its sister impulses and each in its turn sets up new points of reentry If the pattern by which these multiple impulses

When

tern in

The multiple re-entries are sustained by subsequent impulses discharged by the dominant pacemaker or by impulses arising in multiple ectopic foci The main proponents of this theory at the present time are Katz and Pick and Hecht

Focal or multifocal impulse formation —Lewis in his experiments stimulated the auricles with cardiac current to produce atrial flutter and fibrillation Scherf and his associates were the first to induce atrial flutter by the injection of aconitine into the auricular wall By cooling or clamping off the injected auricular appendix they could abolish the aconitine induced flutter but when the clamp was released or the cooling discontinued the flutter reappeared

During aconitine induced flutter Scherf and his colleagues observed that fibrillation could be precipitated by stretching the wall of the auricle and then could be abolished by cooling the site of aconitine application However fibrillation produced by electrical stimulation and topical application of drugs other than aconitine responded differently to cooling This has also been noted by other investigators as will be discussed below Scherf has concluded from these and other observations that flutter probably results from rapid impulse formation in a single focus while fibrillation is caused by rapid impulse formation in either a single center or in several centers More recently Prinzmetal and his associates have reached much the same conclusion concerning the mechanism of flutter and fibrillation

the above theories

to suggest that both mechanisms—circus movement and focal impulse formation—may cause atrial flutter and fibrillation Brown and Acheson produced atrial flutter by the aconitine method of Scherf and of Prinzmetal and by the method of Ramos and Rosenblueth involving rapid electrical stimulation of the auricles The latter method is thought by its formulators to produce a circus movement Not only did these two types of flutter respond differently to various agents but it was also found that in aconitine induced flutter a secondary mechanism existed simultaneously with the primary flutter This secondary mechanism which seemed able to maintain the flutter even when the aconitine focus was abolished was thought to be due either to another spontaneous ectopic focus or to a circus movement

VENTRICULAR FLUTTER AND FIBRILLATION

Ventricular flutter and fibrillation are without doubt, the most serious of the cardiac arrhythmias The theories relating to their mechanism of production are essentially the same as were described for the corresponding atrial arrhythmias atrial flutter and fibrillation and are involved in the same controversy

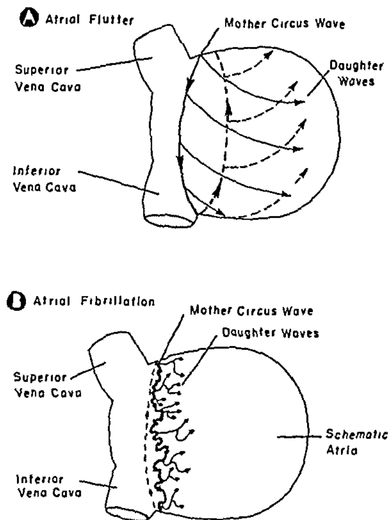


Fig 309 —The circus movement theory of atrial flutter and fibrillation The regularity of *the rhythm and similarity of the atrial flutter* waves observed in the electrocardiogram have been ascribed to the fact that in atrial flutter (A) the mother circus wave enters traverses and *re-enters the same pathway over and over* again while the daughter waves given off by the mother circus wave spread through the atrial myocardium in a constant and orderly way On the other hand in atrial fibrillation (B) the pathway of the mother wave is devious prolonged and variable and the daughter *waves are given off irregularly* and proceed erratically through the atrial muscle Consequently the atria undergo fractional activation and this is responsible for the varying

ence between the circus movement in atrial fibrillation and that in flutter are (1) the more rapid atrial rate and (2) the occurrence of conduction failure in the atrial muscle in atrial fibrillation

mias including atrial fibrillation are significant albeit infrequent *manifestations of digitalis intoxication* In fact atrial flutter resulting from digitalis toxicity is observed very rarely In all of the reported cases of atrial fibrillation or flutter secondary to digitalis intoxication cardiac disease apparently was present in addition

Mechanism of Atrial Flutter and Atrial Fibrillation

The mechanism producing atrial flutter and fibrillation has been the subject of lively controversy for half a decade and the divergence of opinion is just as marked now as ever Although investigations in recent years have provided new and important information concerning the genesis of these two rhythms none of these data incontestably prove or refute any of the theories currently favored These theories are three in number—namely the concept of circus movement the theory of multiple re entries and the theory of focal or multifocal impulse formation

Circus movement —While Lewis and his associates *did not originate the circus movement concept* their extensive investigations seemed to establish it so firmly as the mechanism responsible for atrial flutter and fibrillation that for many years the concept (Fig 309 A and B) was widely accepted and rarely challenged However there were some authorities who regarded this widespread acceptance of the circus movement theory as premature More recently the studies of Scherf and of others have raised new doubt as to the validity of the circus concept and at the same time have tended to favor the mechanism of ectopic focal or multifocal origin of atrial fibrillation and flutter

In essence the circus movement concept supposes that the activation wave called the *mother circus* follows a circular unidirectional and narrow path around the ostia of the two venae cavae During its *course the mother circus gives rise to centrifugally* directed *daughter waves* which spread to all portions of the atria When the mother wave completes its circus movement it immediately re enters the same

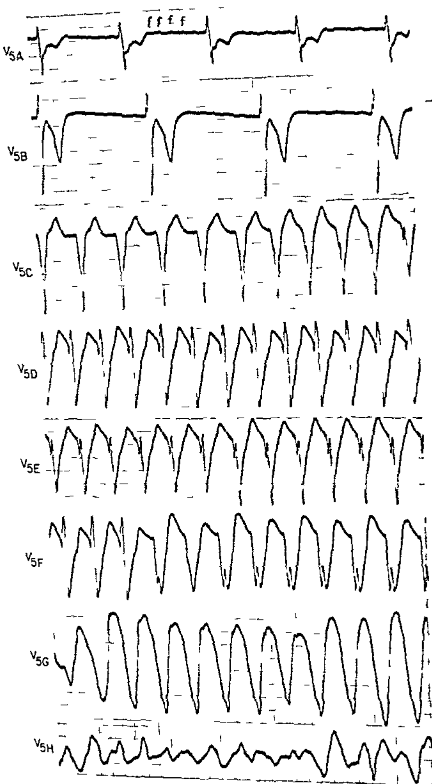
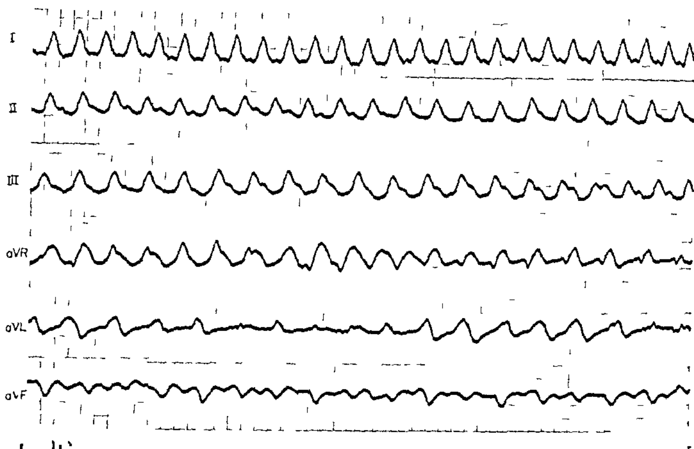


Fig 311 — All of these lead strips are of lead V_5 and they are lettered in the order of their recording In lead V_5 (*A*) fibrillatory waves (*f*) can be seen and therefore atrial fibrillation is present in the atria. The ventricular rhythm is essentially regular despite variations in the f–QRS relationship Thus there must be complete atrioventricular dissociation and block with an idioventricular pacemaker (since the QRS duration exceeds 0 12 second) In lead V_5 (*B*) essentially the same abnormalities are present as in lead V_5 (*A*) except that the ventricular pacemaker has apparently descended lower in the ventricles as evidenced by the fact that the QRS deflections are wider and the ventricular rate is slower than in lead V_5 (*A*) In lead V_5 (*C*) ventricular tachycardia has appeared, and this continues with varying configurations of the ventricular complexes in lead V_5 (*D* and *E*) and in the early portion of lead V_5 (*F*) Later in lead V_5 (*F*) there is marked widening of the ventricular deflections which culminates in the appearance in lead V_5 (*G*) of ventricular flutter Finally in lead V_5 (*H*) ventricular fibrillation supervenes



can be seen and there is atrioventricular dissociation and atrioventricular block. In the remaining leads there is progressive deterioration of the ventricular pacemaker and finally in lead aVF ventricular fibrillation appears. In ventricular fibrillation the ventricular beats are so distorted and appear so irregularly that the tracing presents an entirely chaotic appearance.

Electrocardiographic Features

Ventricular flutter and fibrillation consist of regular undulating continuous waves of large amplitude and marked widening. The normal components of the ventricular complex (QRS and T deflections) cannot usually be distinguished one from the other. During ventricular flutter these waves vary in rate from 180 to 250 per minute and all exhibit about the same configuration. When these waves become more bizarre and variable in appearance and occur with an irregular rhythm, ventricular fibrillation is present. In the terminal stage of ventricular fibrillation the ventricular rate may be slow and quite irregular and the electrocardiogram may show only erratic oscillations of the base line (Figs. 310 and 311).

Ventricular fibrillation is often the terminal cardiac rhythm in patients dying from various causes but especially in death following myocardial infarction or digitalis intoxication. This arrhythmia is frequently observed in Stokes-Adams attacks, when it is most likely to occur during change from a partial to a complete atrioventricular block or during the course of complete atrioventricular block.

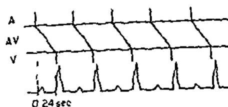


Fig 312 —First-degree atrioventricular block The pur

degree atrioventricular block, or *prolonged atrioventricular conduction* can be diagnosed if every sinus P wave without exception is *followed by a ventricular complex after a P–R interval of 0 21 second or* longer (in adults) (see Figs 312 and 313) This does not hold true when the sinus beat with prolonged atrioventricular conduction is preceded by an ectopic, atrial, nodal, or ventricular extrasystole. Thus a *premature* atrial beat occurring late in the Q–T interval may penetrate the atrioventricular junction deeply without reaching the ventricles The next sinus beat finds the atrioventricular pathways still refractory following concealed conduction of the ectopic impulse and as a result the beat reaches the ventricles only after a long P–R interval Similarly retrograde penetration of the atrioventricular node by an ectopic nodal or ventricular impulse may also be responsible for the slower conduction of the post ectopic sinus beat In the examples cited, atrioventric-

... actually the mechanism producing

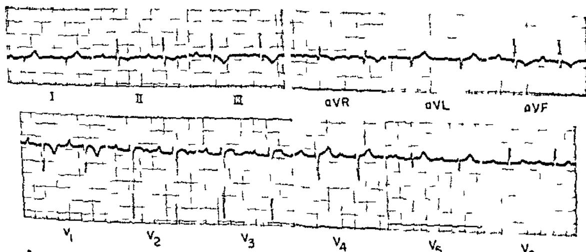
P–R interval of the first postectopic conducted beat

First-degree atrioventricular block is sometimes associated with such marked prolongation of the P–R interval that the latter exceeds the P–P interval in length Thus the sinus beat initiating each ventricular deflection is not the P wave immediately preceding the latter but the P wave before that

Ordinarily in first-degree atrioventricular block the P–R intervals remain constant in length unless the ... rhythm shows some variation in cycle length or

ventricular conducting pathway after the beat terminating a short cycle is responsible for the lengthening P–R intervals) *It need hardly be mentioned that the* 0 21 second value set for the upper limits of the normal P–R interval duration was selected because it applies to the majority of normal subjects On the other hand in occasional normal subjects P–R intervals of 0 22 or 0 24 second may be observed

SECOND-DEGREE ATRIOVENTRICULAR BLOCK.—This type of atrioventricular block is often designated in *complete atrioventricular block* without indicating its degree and, when so used the term "incomplete atrioventricular block" implies that it is second degree block Second-degree atrioventricular block may be



... there is first-degree atrioventricular block ... the patient's age group is less than 0 20 second ... of the ... The

Atrioventricular Block and Sinoatrial Block

Blocked impulse conduction at intra atrial or intra ventricular levels has been described previously in sections dealing with the electrocardiographic patterns of P mitrale and of bundle branch block and diffuse intraventricular block In this chapter only block occurring at or near the sinoatrial and atrioventricular junctions will be considered that involving atrioventricular conduction will receive the greater attention because of the frequency and clinical significance of atrioventricular block and because atrioventricular conduction normal and abnormal has been studied more extensively than sinoatrial conduction However the clinical cardiologist is becoming increasingly aware of the need for recognizing sinoatrial block

ATRIOVENTRICULAR BLOCK

The fact that a sinus beat happens not to be conducted or is conducted slowly by the atrioventricular node does not in itself incriminate atrioventricular block as the causative mechanism to the exclusion of atrioventricular interference There must also be indirect evidence that the refractory period of the atrioventricular junctional tissues is prolonged If for example a ventricular deflection fails to appear after a sinus P wave which falls outside the Q–T interval of the preceding ventricular beat or follows such a P wave by a P–R interval of 0 21 second or longer it can usually be concluded that the refractory period of the atrioventricular junctional tissues extends beyond the Q–T interval—that is beyond the approximate duration of the normal refractory period In either case the fact that prolonged refractoriness of the atrioventricular node is demonstrable substantiates the diagnosis of atrioventricular block

The normal absolute refractory phase corresponds roughly to the initial one half of the Q–T interval and the relative refractory phase of the atrioventricular junctional tissues to the second half of the Q–T interval Since atrial beats (for example premature atrial extrasystoles) occurring shortly after the QRS complex appear within the absolute refractory phase of the atrioventricular node their failure to be conducted is a normal physiologic event Similarly P waves falling near the end of the T wave—that is in the relative refractory period—are conducted either slowly or normally depending on how early or late in this period they arrive at the atrioventricular junction The fact must be kept in mind that the length of the refractory period of the atrioventricular node (and of heart muscle in general) does not remain fixed but varies under the influence of many factors One of the most important of these factors is the relationship between the length of the refractory period and the length of the preceding cycle Within certain limits the longer the preceding cycle length the longer the duration of the refractory period of the beat terminating that cycle and the shorter the preceding cycle the shorter the refractory period

Electrocardiographic Features

Incomplete Atrioventricular Block

The term *incomplete atrioventricular block* implies that some or all of the atrial beats are conducted into the ventricles abnormally slowly Incomplete atrioventricular block can be divided into two types first and second degree atrioventricular block

First degree atrioventricular block —First

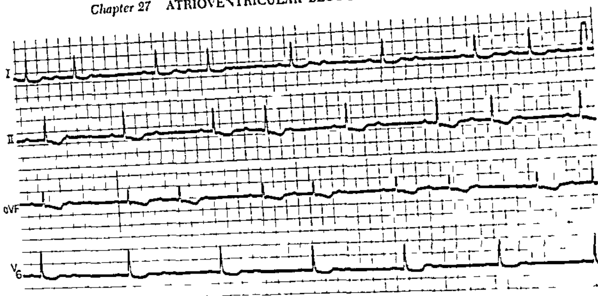


Fig 316 —Incomplete atrioventricular block with periods of 3 2 atrioventricular block of the Wenckebach type and at other times 2 1 block in leads I II and aVF In lead V there is 2 1 atrioventricular block throughout

divided into two subtypes the uncommon type and the common or Wenckebach type

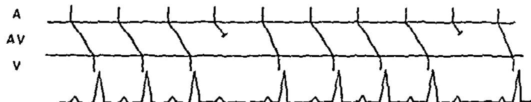
1 The P R intervals of the conducted beats may be prolonged or of normal duration but their lengths remain constant from beat to beat However the conducted beat following the ventricular pause may *sometimes have a shorter* P–R interval than the other beats

2 Ventricular pauses due to blocked atrial beats usually occur at irregular intervals so that the ratio of atrioventricular response varies Sometimes however the ratio of atrial and ventricular beats is fixed

Common or Wenckebach type —In the common type of second-degree atrioventricular block (Figs 314 B 316 and 317) the P–R interval of each successive conducted beat lengthens progressively until finally one atrial beat is blocked This is known as the *Wenckebach phenomenon* The first conducted beat after the pause has a shorter P–R interval (sometimes of normal length) than any subsequent P–R interval and, with its appearance the cycle of lengthening P–R intervals begins once again The electrocardiographic sequence starting with the conducted beat following the ventricular pause and ending with the next blocked atrial beat constitutes a *Wenckebach period* The Wenckebach phenomenon is not a specific manifestation of atrioventricular block alone for it can sometimes result from atrioventricular interference occurring with a rapid atrial rhythm If the atrial rhythm is relatively slow the presence of Wenckebach periods usually indicates depressed atrioventricular conductivity The end result of both of the foregoing mechanisms is that the first conducted atrial impulse of a Wenckebach period arrives at the atrioventricular node during its relative refractory phase Each successive atrial impulse thereafter reaches the atrioventricular node earlier in its recovery period with the result that atrioventricular conduction becomes more and more prolonged as recovery of the junctional tissues becomes progressively less complete Eventually an atrial beat arrives when the junctional tissues are completely refractory and it is blocked Provided the blocked impulse does not penetrate the atrioventricular node deeply the ensuing pause gives the node time to recover more completely For this reason the next atrial beat is conducted more rapidly to the ventricles than those which follow

The explanation usually given for the characteristic features of Wenckebach atrioventricular block is as follows

1 The maximal increment in the P–R interval occurs between the first and second conducted beats following the pause on the other hand although there is gradual lengthening of subsequent P–R intervals the increment in the length of the P–R intervals between consecutive conducted beats becomes less and less The fact that maximal increment in the P–R interval occurs between the first two conducted beats after the blocked atrial beat is probably an expression



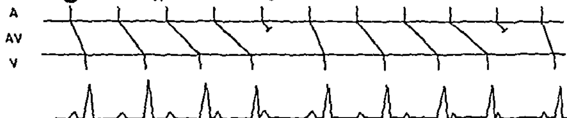
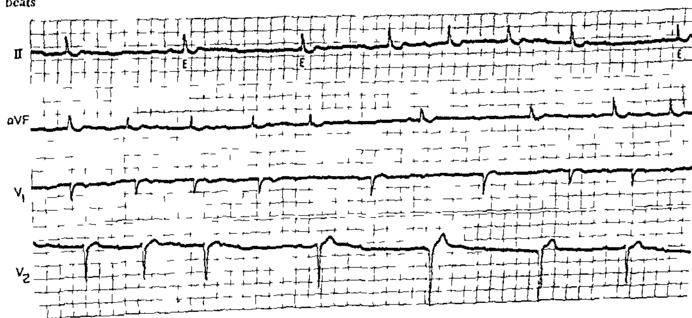


Fig 314 –

trast with the common type of atrioventricular block showing Wenckebach periods (B) In B the fifth and tenth P waves are not conducted into the ventricles Just as in the uncommon type of atrioventricular block the P–R interval of the first conducted beat after the pause is shorter than the P–R interval of all subsequent conducted beats The difference between the two types of incomplete atrioventricular block lies in the fact that in the common type of atrioventricular block there is gradual lengthening of the P–R intervals of subsequent conducted beats until finally one atrial beat fails to emerge from the atrioventricular node The maximal increment in the P–R interval occurs between the first and second conducted beats following the pause All subsequent conducted beats show a diminishing increment in the P–R interval length Consequently the R–R intervals of the conducted beats form what is known as *Wenckebach periods* A Wenckebach period consists of a sequence of conducted beats starting with the first conducted beat following the ventricular pause and ending with the next blocked atrial beat In such a sequence the first R–R interval is the longest shorter and so on The R–R intervals all exceed the P–P cycle length by the in consecutive conducted beats while the ventricular pause due to the blocked s by the decrement in the P–R intervals between the two conducted beats con *taining the pause*

Fig 31ᵏ interval of tricular co the remain beats



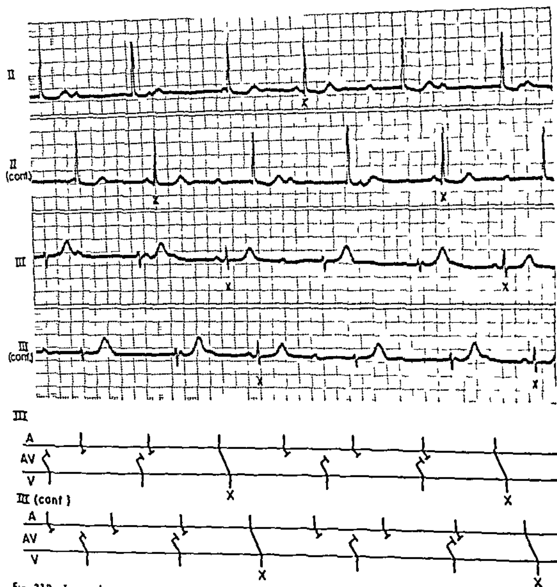


Fig 318 —Incomplete atrioventricular block with 4 1 atrioventricular response and atrioventricular nodal escape
In these continuous lead strips of leads II and III the *ventricular complexes* (*X*) are produced by conducted sinus im
pulses while all other ventricular deflections are atrioventricular nodal escape beats Thus for every one ventricular de
flection produced by a conducted beat there are four P waves Therefore 4 1 incomplete *atrioventricular block* is pres
ent with *atrioventricular nodal escape* *Notice the different QRS configuration* but normal duration of the atrioventricu
lar nodal escape beats as compared with the conducted sinus beats. In the diagram of the two continuous strips of lead
III no attempt has been made to show the variation in depth *of penetration* of the *atrioventricular junctional tissues* by
successive sinus impulses Retrograde atrioventricular block must be assumed to be present to account for the failure of
the retrograde impulses of the first third fifth and seventh nodal escape beats to be transmitted *to the atria before acti*
vation of the latter by sinus impulses

of the parallel relationship existing between the refractory period of the atrioventricular junctional tissues and the length of the preceding R–R cycle. The first conducted beat after the pause follows the longest R–R cycle and therefore induces the greatest increase in the length of the refractory period of the atrioventricular node—hence the maximal increment occurs in the P–R interval of the second conducted beat. The R–R cycles shorten subsequently because the increments in atrioventricular conduction time become less and less owing to the shortening cycle lengths. Actually, the shortening of the R–R cycles of the conducted beats has two opposing effects on the degree of refractoriness of the atrioventricular pathway—namely, a shortening effect due to the shorter cycle lengths per se and a lengthening effect consequent to the increasing fatigue or less complete recovery of the junctional tissues.

2. It follows from the above that each consecutive R–R interval of a Wenckebach period is equal in length to the P–P interval plus the increment in the P–R interval between the two successive conducted beats. On the other hand, the R–R interval containing the blocked P wave equals in length two P–P cycles

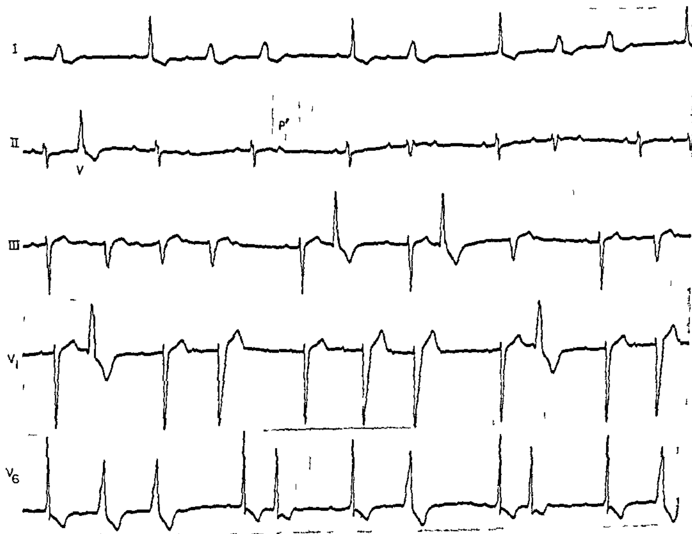


Fig. 317.—Three to two and 4:3 Wenckebach incomplete atrioventricular block with left bundle branch block in the ventricular beats following the first conducted beat after the pause. In lead III, at the beginning of the lead strip, a period of 5:4 incomplete atrioventricular block demonstrates Wenckebach periods. Thus, the first conducted beat has a shorter P–R interval than the following conducted beats; there is progressive lengthening of the P–R intervals of the conducted beats; the R–R cycle lengths decrease progressively. The first conducted beat after the pause shows a

ing a left bundle branch block type of configuration. In lead II, the P wave labeled P′ may be an atrial extrasystole but is probably a sinus P wave which is slightly distorted by artifact.

I

II

aVF

V_1

V_6

Fig

mon bu...



Fig 322 —Complete atrioventricular block with idioventricular pacemaker In contrast with the preceding figures this electrocardiogram shows abnormally widened and deformed QRS deflections Since there is complete dissociation of the faster atrial rhythm and slower ventricular rhythm, complete atrioventricular block must be present and the pacemaker producing the ventricular rhythm must be located in the ventricles below the bifurcation of the common bundle Note also that there is ventriculophasic sinus arrhythmia, as indicated by the measured P–P cycles in lead V

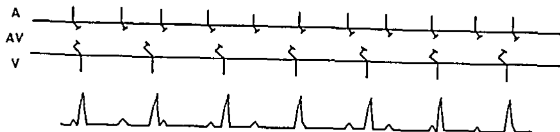


Fig 319 —Third degree or complete atrioventricular block with atrioventricular nodal pacemaker for the ventricles and ventriculophasic sinus arrhythmia As a general rule in ventriculophasic sinus arrhythmia the P–P cycles containing a ventricular beat *are shorter than the P–P cycles not containing a QRS complex* It is thought that mechanical ventricular systole following shortly after the appearance of the electrocardiographic QRS complex causes in some way slightly premature discharge of the sinus pacemaker so that the P–P cycle is shortened This same phenomenon can be seen in incomplete atrioventricular block and in atrial flutter and paroxysmal supraventricular tachycardia with other than 1 1 atrioventricular response

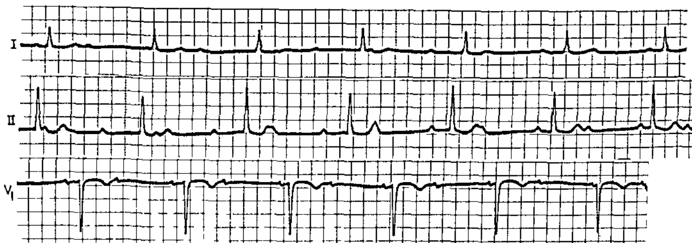


Fig 320 —Complete atrioventricular blo
lar rhythm Thus sinus P waves can be sp

The pacemaker producing the ventricular rhythm must be
ce the *QRS deflections are not widened to 0 12 second*

minus the decrement in the P–R interval between the conducted sinus beats preceding and following the pause

3 The Wenckebach phenomenon therefore consists of recurring series of progressively shorter ventricular cycles separated by long cycles The long cycles or pauses are usually shorter than two short cycles or two *P–P intervals while the first cycle after a pause* is longer than the last cycle preceding a pause

4 The uncommon and common types of second degree atrioventricular block seldom change from one to the other The common or Wenckebach form tends to occur as a temporary complication of digitalis intoxication or as the result of acute damage to the atrioventricular node as in myocarditis or infarction The uncommon form is likely to persist more or less indefinitely and to eventuate in complete or almost complete *atrioventricular* block (Fig 318)

Complete (Third-Degree) Atrioventricular Block

In complete (third degree) atrioventricular block (Fig 319) the atria beat with an independent rhythm and at a faster rate usually than the ventricles The latter respond to a pacemaker *located* somewhere distal to the site of block To facilitate discussion the atrial rhythm will be assumed to be sinus rhythm in the following paragraphs although actually complete atrioventricular block may occur with any type of atrial mechanism The distinctive feature of complete atrioventricular block is that the sinus P waves and the QRS complexes may be spaced off regularly but the P P intervals are usually shorter than the R–R intervals and the P waves and QRS complexes bear no fixed relationship to one another Thus the relative positions of the atrial and ventricular

containing QRS complexes gen

P-]
era
tric...
ventricular cont ... on stimulus form ...
sinus node is pe
terial vagal pres
the influence o ...
the right auricle An additional factor may be changes in the blood supply of the sinus pacemaker owing to mechanical ventricular systole The changing length of the sinus cycles called *ventriculophasic sinus arrhythmia* may
tachycardia or
ular response ...
lar dissociation Rarely the relationship described above is reversed, so that the P–P intervals not containing a QRS complex are shorter than those which do (Fig 323)

Clinical Aspects of Atrioventricular Block

Atrioventricular block of any degree may occur in inflammatory or degenerative diseases of the atrioventricular node or as a manifestation of digitalis quinidine or pronestyl effect. Sometimes it accompanies certain congenital cardiac anomalies particularly high ventricular septal defects and patent atrioventricularis communis Stokes Adams attacks may be produced during transition from an incomplete to a complete atrioventricular block by slowing of the ventricular ... cardiac standstill or by the appear...

... marked slowing of the ventricular rate owing ... shift of the ventricular pacemaker to an area of low rhythmicity (Fig 311)

SINOATRIAL PAUSE SINOATRIAL ARREST AND SINOATRIAL BLOCK

Sinoatrial Pause and Sinoatrial Arrest

Sinoatrial or sinus pause (Fig 324 A) entails failure of the sinus node to discharge one or more impulses Since sinus discharge per se produces no electrocardiographic deflection sinus pause is manifested only indirectly by the unexpected absence of one or more atrial P waves Obviously sinus initiated QRS–T deflections are likewise absent during the pause Ordinarily an atrioventricular nodal pacemaker becomes active fairly promptly and takes over the ventricular rhythm if the sinus pause is prolonged. In the depressed heart the escape rhythm may originate in the ventricle or there may be uniform failure of all pacemakers with resulting cardiac asystole This is almost invariably associated with sinoatrial arrest in which impulse formation in the sinus node presumably ceases completely for a prolonged period Whether or not the outcome is fatal to the patient is determined almost entirely by the activity or inactivity of the secondary cardiac pacemakers Sinus pause may follow an ectopic beat which is conducted retrograde to prematurely discharges and as a consequence depresses the sinus node and not infrequently it is observed after offset of a paroxysmal tachycardia Following some paroxysmal tachycardias —particularly if there is severe underlying cardiac disease—the sinus node depression induced by the ectopic impulses may be so severe as to lead to sinus arrest Sinus pause occurs most commonly and in its most benign form in patients with vagotonia It can be differentiated from sinoatrial block (Fig 324 B) by the fact that the duration of the pause is not a multiple of the usual sinus P–P interval but is widely variable

Sinoatrial Block

In sinoatrial block (Fig 324 B) the excitation impulse forms in the sinoatrial node just as normally is the case but its conduction to the atria is blocked in the sinoatrial junction Thus a P–QRS–T complex does not appear for one or more cycles Typically the R–R interval containing the pause is *exactly* double (or exactly some other multiple of) the interval between two consecutive conducted sinus beats Actually the length of the pause is usually slightly shorter than an exact multiple of the basic sinus cycle because the sinus impulse following the pause is conducted more rapidly through the sinoatrial junction into the atria than is the impulse immediately preceding the pause This is a manifestation of the Wenckebach phenomenon which was previously described in terms of atrioventricular conduction In fact the P–P intervals in sinoatrial block sometimes form Wenckebach periods (Fig 324 C) Sinoatrial block can be diagnosed only when it is a second degree block of either the Wenckebach type or the type in which the pause is a multiple of the sinus cycle In the presence of sinus arrhythmia the diagnosis of sinoatrial block cannot be substantiated A nonconducted atrial extrasystole almost obscured by

beats are constantly changing Occasionally, however the atrial rate may be so nearly a multiple of the slower ventricular rate that the P–QRS relationship remains virtually constant and this causes the atrioventricular block to appear incomplete

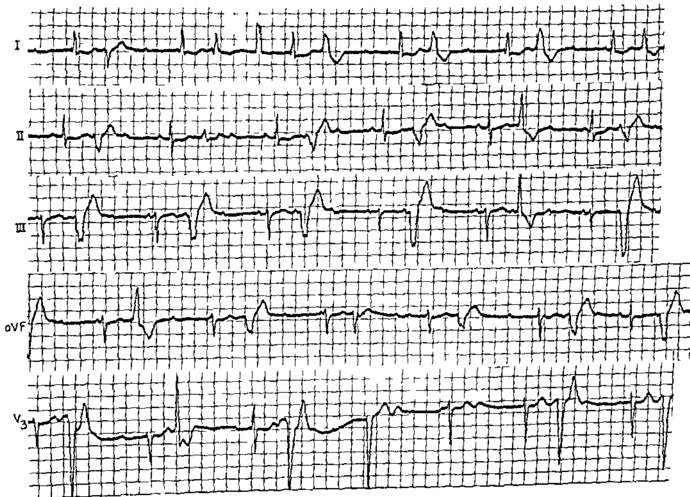
The approximate location of the ventricular pacemaker with respect to the normal conducting pathways is of more than esoteric interest to the clinician If the rhythm center is situated above the bifurcation of the common bundle (or in the atrioventricular junctional tissues below the level of the block) the QRS complexes are essentially normal in appearance and duration When the ventricular rhythm originates below the bifurcation of the common bundle the excitatory impulse spreads in an abnormal fashion through the ventricles The more distal the pacemaker is located in the conduction system the more aberrant the spread of excitation and the greater the deformity and widening of the idioventricular beats

In complete atrioventricular block a ventricular pacemaker in the atrioventricular node or common bundle produces a regular ventricular rhythm with a rate of about 40 beats per minute, while rhythms emanating from more distant ventricular foci tend to be much slower The most significant fact clinically is this the lower in the conducting system is the pacemaker the slower the ventricular rhythm and the greater the possibility that the pacemaker may fail with resulting ventricular standstill With idioventricular centers located above the bifurcation of the common bundle there is much less likelihood of this occurring (Figs 320 and 321)

Ventriculophasic Sinus Arrhythmia in Atrioventricular Block

In complete and incomplete atrioventricular block a frequent finding is variation in the lengths of the

Fig 323 –Complete atrioventricular block with a ventric... originating above the bifurcation of the com... Note that despite the occurrence of extra... quite regular There is a tendency in this record ... than those P–P cycles not enclosing ventricular beats



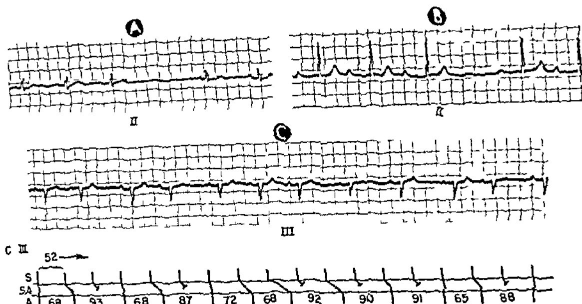
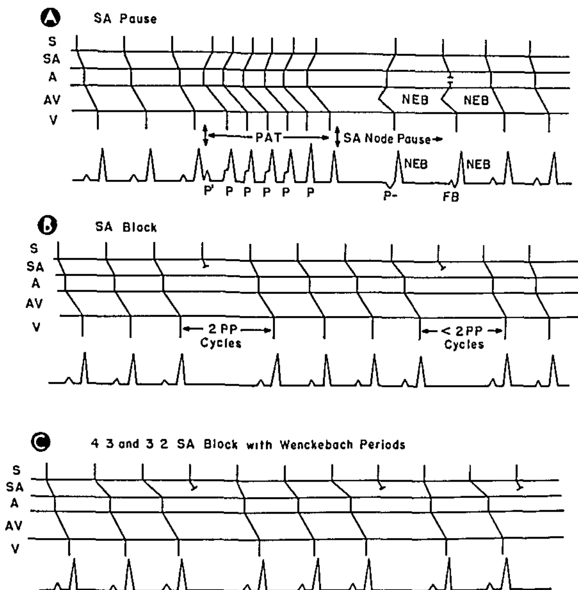


Fig 325 —Incomplete sinoatrial block A a short strip of lead II with a pause in the sinus rhythm due to sinoatrial block The P–P interval containing the pause is twice the P–P interval of the preceding cycle B a strip of lead II from another patient In this case the pause in the atrial rhythm is less than two times the basic P–P cycle length This may represent the effect of differences in the rate of sinoatrial conduction of the sinus impulses preceding and following the pause C a long strip of lead III obtained from still another patient In this instance there appears to be incomplete sinoatrial block with ratios of sinoatrial conduction varying between 4 3 3 2 and 2 1 The period of 4 3 sinoatrial block appears in about the middle of the lead strip and the P–P cycle lengths in this period show Wenckebach periods Undoubtedly some degree of sinus arrhythmia is present but in the diagram of lead III the cycle length of sinus node discharge is represented as having a constant value of 0.52 second



of the rhythmicity of the pacemaker there ensues a long pause before the sinoatrial node initiates its first impulse after the tachycardia However before this impulse can be formed and discharged from the sinoatrial node a retrograde atrioventricular nodal impulse is able to spread through the atria to produce a retrograde P wave (*P−*) to penetrate the sinoatrial junctional tissues and to discharge prematurely the forming sinus impulse Thus the first atrioventricular nodal escape beat (*NEB*) following the bout of paroxysmal tachycardia captures ventricles atria and sinoatrial node The second atrioventricular nodal escape beat initiates ventricular excitation but as it passes in a retrograde direction it encounters the descending sinus impulse in the atria and so an atrial fusion beat (*FB*) is recorded Following this the sinoatrial node takes over once again as dominant pacemaker Thus the sinoatrial pause extends from the last P

tion of the two sinus beats containing the blocked sinus impulse In **C** a more complicated form of sinoatrial block namely incomplete sinoatrial block with Wenckebach periods is illustrated This type of sinoatrial block can be recognized by the regularly recurring Wenckebach periods in the atrial rhythm Thus the P–P cycle length decreases progressively until finally a long pause ensues Then the cycle of shortening P–P cycle lengths is repeated The explanation for the Wenckebach periods in this form of sinoatrial block is exactly the same as was given in the corresponding type of atrioventricular block except that in this instance the variation in the rate of impulse conduction occurs in the sinoatrial junctional tissues

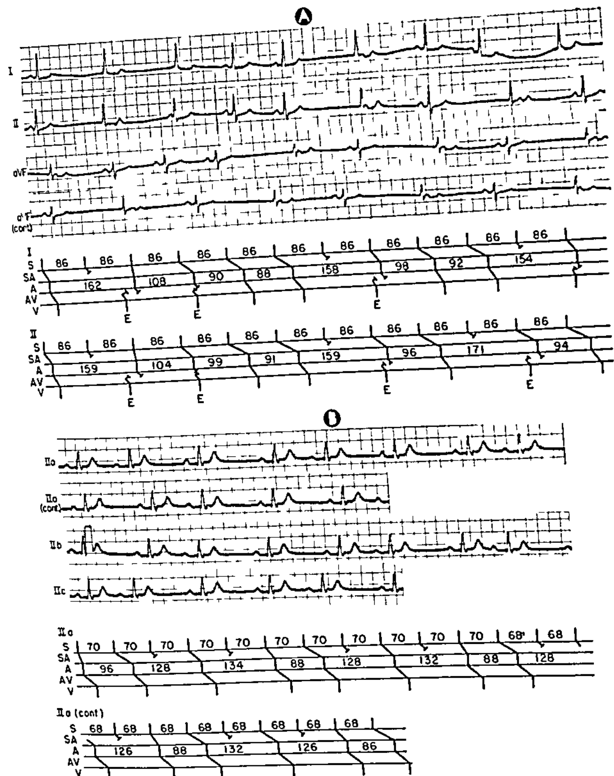


Fig 327 —A strips of leads I and II and two continuous lead strips of lead aVF showing varying degrees of Wenckebach sinoatrial block with atrioventricular nodal escape beats (*E*) Note the regularly recurring pattern of decreasing P–P cycle lengths during the periods of 5 4 and 4 3 sinoatrial block (see diagrams of leads I and II) B lead strips of lead II recorded from a different patient and showing what probably is 2 1 and 3 2 Wenckebach sinoatrial block (see diagrams of the two strips of lead II *a*) In addition, there is first-degree atrioventricular block

Fig 326 —Varying degrees of sinoatrial block with atrioventricular nodal escape In lead V_3 the first second fourth sixth and seventh ventricular complexes are atrioventricular nodal escape beats but the P waves preceding them are sinus P waves (which are labeled *P* in lead V_4) The third and fifth QRS complexes are conducted beats The longer P–P cycles (2 00 seconds) in this lead strip are almost twice the shorter P–P cycles (1 03 and 1 05 seconds) indicating 3 2 sinoatrial block In the last portion of the lead there is 2 1 sinoatrial block Two continuous strips of lead V_4 show predominantly 2 1 sinoatrial block with the third fourth and fifth ventricular complexes being atrioventricular nodal escape beats (*E*) and the first second and sixth ventricular beats being conducted beats In the second strip of lead V_4 the longer P–P cycles are slightly shorter than twice the shortest P–P cycles but this can be attributed to variations in sinoatrial conduction of the sinus impulse preceding and the sinus impulse following the blocked beat The two strips of lead V_4 are presented in diagram form to demonstrate the mechanisms responsible for the electrocardiographic findings

PART IV

Other Conditions Affecting the Electrocardiogram

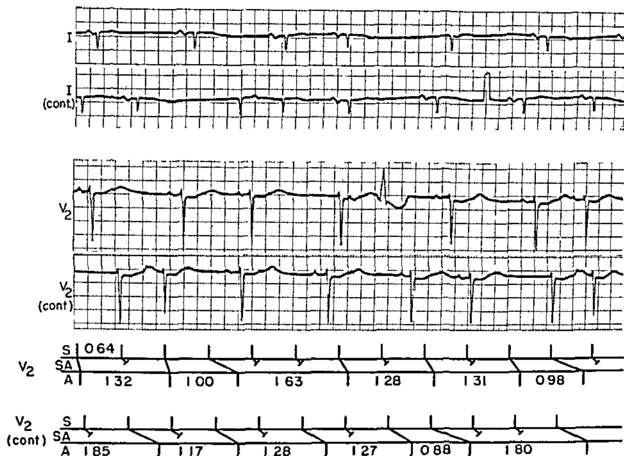


Fig 328 —Varying degrees of sinoatrial block The two continuous strips of lead V_2 are diagramed below the electrocardiogram If it is assumed that the sinoatrial conduction times of the sinus impulses producing the second third and fourth P waves in the second strip of lead V_2 are probably the same then the calculated cycle length of sinus node discharge is approximately 0 64 second or one half the length of the P–P cycles of the aforementioned P waves On spacing out this interval with reference to
block is demonstrated Note that the peri
consecutive sinoatrial node discharges A

a T wave may simulate sinoatrial block and so a search should always be made for an ectopic P wave preceding the pause (Figs 325–328)

Clinical Aspects of Sinoatrial Pause Sinoatrial Arrest and Sinoatrial Block

Sinus node pause sinus arrest and sinoatrial block may be observed (*a*) in normal subjects with increased vagal tone or during infectious diseases (*b*) in normal subjects with a hypersensitive carotid sinus (*c*) in patients with sinoatrial node involvement by coronary artery disease or myocarditis or (*d*) as toxic manifestations of excess digitalis or quinidine or of hyperkalemia It has been stated that sinoatrial arrest when due to toxic drug effects may signify impending cardiac arrest

For a detailed discussion of the effects of the drugs digitalis quinidine and pronestyl and of hyperkalemia on the heart and the electrocardiogram see Chapter 28

Digitalis, Quinidine, and Pronestyl, Electrolyte Imbalance

EFFECT OF DIGITALIS ON IMPULSE FORMATION AND TRANSMISSION

Digitalis Action

A comprehensive discussion of the effects of digitalis on the rhythm of the heart beat is beyond the scope of this text Instead, the actions of digitalis are to be sketched only briefly in the following paragraphs but the references listed in the bibliography may be consulted for a more detailed presentation of this subject

The influence of digitalis on cardiac rhythmicity excitability and conductivity is mediated through *two distinct actions*—direct and indirect In the *direct action* the digitalis acts directly on heart muscle and produces myocardial depression qualitatively similar to but quantitatively less intense than that produced by quinidine In the *indirect action* the digitalis causes vagal stimulation Thus its indirect action is exerted through the intermediate agency of the vagal nerves and is equivalent in effect to increased vagal tone Although the vagus nerves were not previously thought to extend as far as the ventricles vagal stimulation recently has been demonstrated to influence the ventricular rhythm

The actions of digitalis at different levels in the heart and their clinical implications are outlined below

Sinoatrial and Atrioventricular Nodal Pacemakers

The rhythmicity of these centers is inhibited by both direct and indirect actions of digitalis This is exemplified by sinus bradycardia by *sinus arrhythmia*, or by the wandering supraventricular pacemaker which sometimes appears during digitalis therapy Fluctuating degrees of sinus node inhibition may from time to time permit an atrioventricular nodal pacemaker to escape at a *slightly faster rate* and intermittent complete atrioventricular dissociation may appear

Sinoatrial and Atrioventricular Conduction Pathways

The direct and indirect actions of digitalis have a synergistically depressive effect on sinoatrial and atrioventricular conductivity and prolong the refrac

fibrillation the depressant effect of digitalis on atrioventricular conduction is therapeutically desirable while under other circumstances it *may signify* digitalis toxicity

Atrial Myocardium

The influence of digitalis in the atria is complex if not actually unpredictable to a certain degree Sometimes the direct and indirect actions of digitalis augment at other times antagonize each other

In general the effects of digitalis *on the atria* are as follows

Excitability—Both directly and indirectly digitalis depresses the excitability of atrial muscle and raises its *excitability threshold to* stimulation

Refractory period—The refractory period of atrial

ting

by

Digitalis, Quinidine, and Pronestyl, Electrolyte Imbalance

EFFECT OF DIGITALIS ON IMPULSE FORMATION AND TRANSMISSION

Digitalis Action

A COMPREHENSIVE DISCUSSION of the effects of digitalis on the rhythm of the heart beat is beyond the scope of this text Instead, the actions of digitalis are to be sketched only briefly in the following paragraphs but the references listed in the bibliography may be consulted for a more detailed presentation of this subject.

The influence of digitalis on cardiac rhythmicity excitability and conductivity is mediated through two distinct actions—direct and indirect In the *direct action* the digitalis acts directly on heart muscle and produces myocardial depression qualitatively similar to but quantitatively less intense than that produced by quinidine In the *indirect action* the digitalis causes vagal stimulation Thus its indirect action is exerted through the intermediate agency of the vagal nerves and is equivalent in effect to increased vagal tone Although the vagus nerves were not previously thought to extend as far as the ventricles vagal stimulation recently has been demonstrated to influence the ventricular rhythm

The actions of digitalis at different levels in the heart and their clinical implications are outlined below

Sinoatrial and Atrioventricular Nodal Pacemakers

The rhythmicity of these centers is inhibited by both direct and indirect actions of digitalis This is exemplified by sinus bradycardia by sinus arrhythmia, or by the wandering supraventricular pacemaker which sometimes appears during digitalis therapy Fluctuating degrees of sinus node inhibition may from time to time permit an atrioventricular nodal pacemaker to escape at a slightly faster rate and intermittent complete atrioventricular dissociation may appear

Sinoatrial and Atrioventricular Conduction Pathways

The direct and indirect actions of digitalis have a synergistically depressive effect on sinoatrial and atrioventricular conductivity and prolong the refractory period of the junctional tissues According to the dosage level digitalis can produce all degrees of sinoatrial and atrioventricular block In atrial flutter and fibrillation the depressant effect of digitalis on atrioventricular conduction is therapeutically desirable while under other circumstances it may signify digitalis toxicity

Atrial Myocardium

The influence of digitalis in the atria is complex if not actually unpredictable to a certain degree Sometimes the direct and indirect actions of digitalis augment at other times antagonize each other

In general the effects of digitalis on the atria are as follows

Excitability —Both directly and indirectly digitalis depresses the excitability of atrial muscle and raises its excitability threshold to stimulation

Refractory period —The refractory period of atrial muscle is greatly shortened by the vagal stimulating action of digitalis and also but to a lesser extent by its direct muscular effect

Conductivity—Digitalis seems to depress the conductivity of atrial muscle However Scherf believes that the direct depressant action of digitalis on atrial conductivity is not yet supported by adequate proof

Conversion of atrial flutter—Ordinarily the indirect action of digitalis tending to shorten the refractory period of the atrial muscle dominates its depressant effect on conductivity and for this reason digitalis often converts atrial flutter to fibrillation In the occasional instances in which the reverse holds true—that is the direct action being predominant—digitalis may cause atrial flutter to revert to sinus rhythm

Ventricular Myocardium

Although vagal fibers apparently reach the ventricular myocardium the effects of vagal stimulation on the ventricles are rarely seen and when present are of minor importance Consequently the indirect action of digitalis in the ventricles is negligible and the direct action causing depressed conductivity and excitability is predominant It has been stated that if digitalis lessens conductivity to a greater extent than it depresses excitability it may convert ventricular tachycardia to ventricular fibrillation As far as is known digitalis does not affect the conductivity of the intraventricular conducting pathways at least to a degree demonstrable in the electrocardiogram

Interrelationship of Digitalis and Potassium

Apparently both the toxicity of digitalis and perhaps even more fundamentally its effect on the failing heart are involved in the complicated problem relating to the potassium metabolism of the myocardial cell It is thought that the intracellular potassium ions surrounding the paired actin and myosin molecules prevent these proteins from uniting owing to the mutually repelling action of ions having the same electrical charge Excitation causes the cell membrane to become permeable to potassium ions which diffuse out of the cell into the potassium poor extracellular fluid Loss of their potassium ion atmosphere permits actin and myosin to unite and after absorbing adenosine triphosphate the resulting protein complex contracts Thus the rapid migration of potassium ions to and fro across the cell membrane constitutes an important connecting link between electrical and mechanical cardiac systole and diastole

Digitalis has been noted to improve the contractile dynamics of actomyosin solutions Horvath and his associates feel that they have demonstrated a direct effect of digitoxin on the polymerization of actin However it has been pointed out that conceivably these observed effects of digitalis may also reflect the effects of digitalis induced alterations in the intracellular potassium ion concentration The following additional observations by various investigators seem to imply a significant relationship between digitalis and the intracellular potassium of the heart

1 Toxic doses of digitalis are said to liberate potassium from heart muscle From a clinical standpoint this is the explanation presumably for the reported effect of the toxic dose of digitalis in abolishing the electrocardiographic findings of hyperkalemia or lessening their prominence in patients with acute renal insufficiency Several patients so treated and maintained on high doses of digitalis survived episodes of severe hyperkalemia

2 Digitalis in therapeutic doses has been claimed by many investigators to increase the intracellular potassium ion concentration of the heart but others have reported that digitalis lowers or has no effect on the potassium content of heart muscle Clarke and Mosher found the potassium content of heart muscle obtained at autopsy to be lower than normal in undigitalized cardiac patients and normal or near normal in those who had received digitalis prior to death In parallel with this observation there is evidence suggesting that during the development of congestive heart failure there is a loss of body potassium with a concomitant reduction in the cardiac intracellular potassium concentration especially in those heart chambers which are failing It has been proposed without conclusive proof that the beneficial effect of digitalis on the failing heart emanates from its action in restoring the heart muscle potassium to normal levels Experimentally digitalis in nontoxic doses has been found to prolong the life of adrenalectomized animals This observation gives some substance perhaps to the belief that digitalis acts to maintain potassium within the heart cell

3 Withdrawal of potassium from the body by dialysis or by mercurial diuresis has been shown experimentally and clinically to increase the sensitivity of the heart to digitalis Thus the removal of potassium from the body may precipitate digitalis intoxication despite an unchanged schedule of maintenance digitalis therapy Conversely most of the ectopic arrhythmias resulting from digitalis intoxication respond readily to potassium therapy Sensitivity of the heart to digitalis is decreased and resistance to the induction of digitalis intoxication increased by the presence of elevated body potassium levels

4 It has been shown that stimulation of the vagus nerve causes a loss of potassium from the heart As was previously stated the vagal stimulating effect of digitalis is largely confined to the atria and to the junctional tissues of the sinoatrial and atrioventricular nodes In view of the preceding facts it is of interest that Sherrod noted significant potassium depletion in atrial muscle but no significant change in the potas

Digitalis Effect on the Electrocardiogram

Pa ... val S-T segment and T wave The magnitu

TABLE 29 —DISTURBANCES OF IMPULSE FORMATION AND CONDUCTION DUE TO DIGITALIS

	Disturbance of Impulse Formation	Disturbance of Impulse Conduction
Excessive Digitalis Effect	1 Depressed rhythmicity of the sinoatrial node leading to sinus bradycardia wandering supraventricular pacemaker sinus arrhythmia and atrioventricular nodal escape with atrioventricular dissociation and a ventricular rate less than 60 beats per minute	1 First or second-degree atrioventricular block.
	2 Atrioventricular nodal rhythm with atrioventricular dissociation a ventricular rate faster than 60 beats per minute and retrograde atrioventricular block (occasionally with incomplete forward atrioventricular block)	2. Sinoatrial block with atrioventricular nodal escape
	3 ... beats ... beats) ... digita... gin as evidenced by their constant coupling inter als	3 High degree of atrioventricular block in the presence of atrial fibrillation as evidenced by a slow almost regular ventricular rhythm with frequent atrioventricular nodal escape
Therapeutic implications	Digitalis can be continued cautiously preferably at a reduced dosage	
Digitalis Intoxication	The appearance of any of the following abnormalities in the electrocardiogram	
	1 Premature ectopic ventricular beats giving rise to a bigeminal rhythm VPCs of multifocal origin or paired bidirectional VPC's or VPCs occurring in short runs of consecutive beats	1 Complete atrioventricular block
	2 Paroxysmal ectopic tachycardia Paroxysmal atrial tachycardia with atrioventricular block Paroxysmal atrioventricular nodal tachycardia atrioventricular dissociation and retrograde atrioventricular block Simultaneous paroxysmal atrial and atrioventricular nodal tachycardias with complete atrioventricular dissociation Alternating bidirectional tachycardia Paroxysmal ventricular tachycardia	
	3 Paroxysmal atrial or ventricular flutter or fibrillation	
Therapeutic implications	Digitalis should be discontinued immediately Oral or intravenous potassium chloride is the treatment of choice Quinidine and pronestyl should not be given.	Therapy is the same as that used in complete atrioventricular block due to other causes

For all intents and purposes the conduction disturbances produced by digitalis ... limited to the sinoatrial and atrioventricular ... conducting pathway ... ventricular conduction abnormalities due to digitalis have not been reported as far as the authors can determine

TABLE 30 – Effects of Vagal Stimulation, Exercise and Drugs on Ectopic Tachycardias, Flutter and Fibrillation

	Supraventricular Tachycardia (SVT) (Atrial or Atrioventricular Nodal) — Without Atrioventricular Block	Supraventricular Tachycardia (SVT) — With Atrioventricular Block	Atrial Flutter (AFl)	Atrial Fibrillation (AF)	Ventricular Tachycardia (VT)
Carotid sinus stimulation	1 Terminates SVT in 65% of cases 2 Alternatively no effect	1 Rarely terminates SVT with atrioventricular block 2 Usually increases degree of atrioventricular block and slows ventricular rate	1 Usually temporarily increases degree of atrioventricular block and slows ventricular rate to ½, ⅓ etc of the original rate Despite continued vagal stimulation there is usually an irregular return of ventricular rate to its former level 2 Occasionally increases the atrial rate of AFl or converts flutter to fibrillation	Increases degree of atrioventricular block with irregular slowing of ventricular rate	1 Usually no effect 2 Rarely decreases ventricular rate
Exercise	No effect	May decrease degree of atrioventricular block and increase ventricular rate	Either no effect or decreases degree of atrioventricular block and increases ventricular rate	Decreases degree of atrioventricular block temporarily and temporarily increases ventricular rate Apparently these effects can occur despite adequate digitalis medication	No effect
Increased vagal tone produced by parasympathomimetic drugs	Often terminates SVT	Same effects as carotid sinus stimulation but more marked and long lasting	Same effects as carotid sinus stimulation but more marked and long lasting	Same effects as carotid sinus stimulation although more marked and longer lasting	1 No effect 2 (?) May rarely convert VT to ventricular fibrillation
Digitalis	Usually terminates SVT	1 When digitalis toxicity is not a causative factor digitalis terminates SVT with atrioventricular block in less than 50% of the cases 2 In over 50% of the cases the degree of atrioventricular block is increased and ventricular rate is slowed	1 Sometimes converts AFl to sinus rhythm directly 2 Usually converts AFl to atrial fibrillation which in about 65% of such cases reverts to sinus rhythm on cessation of digitalis 3 Increases degree of atrioventricular block and slows ventricular rate	1 Same effects as parasympathomimetic drugs 2 Occasionally reverts AF directly to sinus rhythm particularly if the AF is acute or paroxysmal rather than chronic or if AF is secondary to congestive heart failure	Occasionally may terminate VT not due to digitalis intoxication when other measures have failed

[illegible]	[illegible]	[illegible] ... block (5 of 7 cases reported by Lown and Levine)	1 Q [illegible] 30–65% [illegible] 2 [illegible] paroxysmal AFl to sinus rhythm but has little effect in chronic AFl unless used in massive and potentially toxic dose	1 Q [illegible] 80% [illegible] 2 [illegible] efficacy [illegible] AFl	[illegible]
Potassium	May terminate SVT with or without atrioventricular block if the SVT is due to digitalis intoxication		Effects of potassium on AFl and AF have not as yet been well studied but potassium may terminate these atrial arrhythmias when they are due to digitalis intoxication		May terminate VT due to digitalis but is a less successful therapeutical[ly] in VT not due to digitalis excess

At [illegible] two types of ectopic tachycardias in this table both being grouped under the heading [illegible] drug [illegible]

these changes correlates in only a very approximate and *inconstant* way with the amount of digitalis administered For this reason the appearance of digitalis effect does not *augur* digitalis toxicity nor does it indicate that a therapeutic dose of the drug has been given In fact the presence or absence of electrocardiographic digitalis effect affords no proof that a given patient has or has not received digitalis in the recent past This point is illustrated by the occasional patients severely intoxicated with digitalis who display *normal electrocardiograms* and their opposites—that is patients not under digitalis therapy who show T wave and S–T segment changes which mimic digitalis effect but are due to other conditions The electrocardiographic features of *digitalis effect* are as follows

Shortening of the Q–T Interval

[illegible] of the heart so that repolarization of the ventricular wall is accomplished more quickly than normally The shortening of the Q–T interval is of little value clinically in the detection of digitalis effect unless previous electrocardiographic tracings are available for careful measurement and comparison of the Q–T intervals

Changes in the S–T Segment and T Wave

With mounting intensity of digitalis effect the following two changes occur (*a*) The S–T vector representing early repolarization forces tends to increase in magnitude as the T vector representing late repolarization potentials decreases (*b*) The mean S–T vector is directed away from the mean QRS vector while the mean T vector continues to parallel the QRS vector unless digitalis completely reverses the direction of *repolarization* In this *instance* the S–T and T vectors point away from the mean QRS vector

In terms of the electrocardiogram the above orientations of the mean QRS S–T and T vectors relative *to* one another produce the digitalis effect pattern consisting of depressed S–T segments with small upright terminal T waves or inverted T waves in leads registering resultantly positive QRS complexes (see also Chapter 6 p 85)

[illegible] T *changes due* [illegible] leads recording [illegible] *result in the following* manner (*a*) Minor degrees of digitalis effect may cause *simultaneous repolarization* both in the

normal epicardial to endocardial direction and in the *reverse direction* the latter effect being due to more rapid recovery in the subendocardium These two repolarization forces directed oppositely yield a resultant mean T vector of decreased magnitude whose orientation is not markedly altered Because of the shortened Q–T interval depolarization and repolarization tend to overlap so that repolarization forces directed from endocardium to epicardium appear before inscription of the QRS is completed The S–T segment and its point of junction with the ventricular complex (designated J) are displaced downwardly as a result Subsequently the outer layers of myocardium whose recovery process is less affected by digitalis repolarize in the normal direction and a small upright terminal T wave is written (*b*) Often digitalis effect is so intense that the entire ventricular wall repolarizes in a reverse direction—that is from endocardium to epicardium In such a case *the S–T segment is depressed and the T wave is inverted*

Digitalis acts to abolish the subendocardial recovery delay which is present normally and is responsible for the normal ventricular gradient The ventricular gradient becomes smaller as the variations in the duration of the excited state between inner and outer layers of heart muscle decrease with onset of digitalis effect If the action of digitalis in reducing the *ventricular gradient is coupled with another influence* also likely to decrease the gradient—such as exercise ventricular hypertrophy transmural ischemia or hypoxia—S–T and T wave changes which were not present previously may appear Often the S–T segment depression secondary to digitalis is relatively distinctive because of its straight line or downwardly bowed appearance The S–T segment depression caused by other conditions more commonly exhibits upward *bowing of the S–T segment Unfortunately* the configuration of depressed S–T segments varies so widely that it is of little practical value in differentiating the S–T segment deviations of digitalis effect from changes due to other causes It is interesting that the ST–T changes of digitalis can be nullified by potassium salts However intracellular potassium loss cannot be held responsible for the shortening of the Q–T interval observed in digitalis therapy

The paradoxical effects of digitalis on impulse formation and conduction in the heart may be summarized in general terms as follows (see also Tables 29 and 30)

1 The depressant action of digitalis on atrioventricular conductivity in particular is sometimes therapeutically desirable (e g when digitalis is used to slow the ventricular rate in atrial flutter or fibrillation) but at other times is a manifestation of digitalis toxicity Thus the significance of digitalis induced atrioventricular block may depend in a given instance on the therapeutic objective of digitalis administration

2 Digitalis is generally considered to be a cardiac depressant and yet *on occasions it apparently* enhances the rhythmicity of secondary cardiac pacemakers This effect is exemplified by the relatively rapid rate of atrioventricular nodal rhythms with atrioventricular dissociation which sometimes appear as manifestations of digitalis toxicity By the same token in complete atrioventricular block produced by toxic doses of digitalis the rate of the ventricular pacemaker tends to be more rapid than that in complete atrioventricular block not related to digitalis

3 Digitalis toxicity may be manifested by the appearance of rapid ectopic rhythms which under other circumstances are abolished by administration of digitalis Thus digitalis may terminate paroxysmal supraventricular tachycardias atrial flutter and atrial fibrillation Conversely toxic levels of digitalis in the body may produce any of the preceding abnormal rhythms (including paroxysmal ventricular tachycardia)

A clinical observation familiar to all is the varying susceptibility of different patients to digitalis intoxication This can be attributed in some cases to differences in the severity of the underlying cardiac disease to the presence or absence of derangements of body electrolytes (notably potassium sodium calcium *and possibly magnesium) or to the existence* of active myocarditis myocardial ischemia or some other disease process elsewhere in the body However the predilection of other patients to digitalis intoxication is not related to any factors which can be recognized in advance and for which allowances can be made during digitalization Consequently *the effects* of digitalis administration on impulse formation and conduction in the heart are somewhat unpredictable and so digitalization of a patient is in a sense an experiment which should be monitored by the electrocardiogram to avoid digitalis intoxication or to detect it at its earliest stage The extracardiac manifestations of digitalis toxicity will not be described

The terminology used in this text for designating the electrocardiographic manifestations *of digitalis* is as follows (see also Table 29)

Digitalis effect —Digitalis effect may be summarized as follows Digitalis hastens the repolarization process in the ventricular muscle This action is re

flected in the electrocardiogram by a shortening of the Q-T interval by S-T segment deviation, and by T w ... nce or absence or the promi n ... heart muscle

Excessive digitalis effect —This term is applied to electrocardiographic findings which represent undesired or even toxic digitalis effects but are not contraindications to continued cautious administration of the drug

Digitalis intoxication —This is the designation given to those digitalis induced abnormalities of impulse formation or conduction which preclude further digitalis therapy

QUINIDINE AND PRONESTYL

Quinidine and pronestyl (procaine amide) differ in many respects besides their chemical structures but in terms of their effect on the heart and electrocardiogram the similarities of the two drugs far outnumber their dissimilarities Comparison of the clinical value of quinidine and pronestyl has led to some controversy However at present it is believed that at comparable dosages (250 mg capsule of pronestyl = approximately 1 gr of quinidine) quinidine and pronestyl are of equal therapeutic efficacy in most of the arrhythmias excluding chronic atrial fibrillation a ... In these arrhythmias quinidine is more s ...

the same effect on the heart as procaine amide (pronestyl) but is so rapidly hydrolyzed in the body that its effect is quite transient and (b) quinine and atabrine apparently have cardiac effects resembling those of quinidine but less marked

Actions of Quinidine and Pronestyl

Vagal paralysis —Once quinidine or pronestyl has ...

pronestyl can lead to a marked acceleration in the ventricular rate

Decreased conductivity —Both drugs slow conduction recovery and lessen conductivity

Depressed excitability —The excitability threshold for response to stimulation is raised and irritability and excitability is depressed

Slowing of rate of discharge of an ectopic focus —Prinzmetal and his co-workers believe this to be the main antiarrhythmic action of quinidine although it may not be separate and distinct from the two preceding drug effects These investigators also tentatively suggest that the vagolytic action of quinidine may contribute to the depressant action on the ectopic pacemaker Usually the ectopic focus is more sensitive to the depressant effect of quinidine than is the sinoatrial node Therefore when the ectopic pacemaker is slowed by quinidine to a rate below that at which the sinoatrial node is discharging the arrhythmia is supplanted by a normal sinus rhythm The occasional therapeutic failures of quinidine may stem from the fact that in these cases the ectopic focus has a resistance to quinidine equaling or surpassing that of the sinoatrial node

While it is a common assertion that quinidine and pronestyl lengthen the refractory period of atrial and ventricular muscle in actuality neither drug has this effect Quinidine has even been demonstrated in some cases to shorten the refractory period The antiarrhythmic properties of quinidine and pronestyl are derived chiefly from their depressant action on the conductivity and excitability of the heart with perhaps an additional factor—namely their tendency to slow the rate of discharge of the ectopic focus The muscle and conductive tissues of all regions of the heart share to a varying degree the depressant effect of the two drugs—hence the applicability of these drugs to both supraventricular and ventricular arrhythmias (Table 30) However this widespread therapeutic action is accompanied by an equally widespread toxic action of the drugs so that virtually any portion of the electrocardiographic complex may display drug induced abnormalities

The ECG Changes Due to Quinidine and Pronestyl

The following changes are probably within the limits of therapeutic effect

1. Prolongation of the Q–T interval—the earliest manifestation of quinidine effect. This manifestation, although frequently observed during pronestyl therapy, may occasionally fail to be present. Moreover, myocardial disease, which is present more often than not in patients receiving quinidine, also causes Q–T interval prolongation.
2. Some widening and notching of the P waves.
3. Flattening or inversion of the T waves and slight depression of the S–T segments.

The changes which are probably representative of toxic drug effect are the following:

1. Possible increase in the duration of the P–R interval.
2. Slowing of the atrial rate, development of intra-atrial block, and atrial standstill in severe toxicity.
3. Progressive widening of the QRS complex with increasing blood levels of quinidine or pronestyl. Sokolow states that ordinarily the usual dose of 2–3 Gm. of quinidine orally per day does not cause significant widening of the QRS interval. Drug therapy should be stopped if the QRS interval becomes widened by 50% of its control width, and caution should be exercised by recording an electrocardiogram before each dose of the drug when the drug-induced increment in the QRS interval approaches 25% of the control interval. Marked widening of the QRS deflections may precede onset of ventricular tachycardia or ventricular fibrillation.
4. According to Sokolow and others, the appearance of ventricular arrhythmias, such as ventricular extrasystoles and ventricular tachycardia, often represents toxic manifestations of quinidine (and presumably pronestyl).

Just as there is some disagreement over the respective therapeutic efficacies of pronestyl and quinidine, so also there is differing opinion concerning their comparative toxicities. While conclusive proof is lacking, many clinicians are of the conviction that pronestyl is a less toxic agent than quinidine, a view which Scherf does not accept.

ELECTROLYTE IMBALANCE

Only disturbances of body potassium and calcium will be described, since less is known of the cardiac effects of other electrolytes and since in clinical electrocardiography disturbances in body potassium and calcium are relatively frequent occurrences.

Potassium

Ninety-eight per cent of the body potassium resides intracellularly, but routinely it is possible to measure only the venous serum potassium concentration. It is evident that determination of the latter value can have only limited implications in terms of the total body potassium and intracellular potassium concentration because the extracellular potassium constitutes such a small fraction of the total. Hyperkalemia (hyperpotassemia) is said to be present when the serum potassium level exceeds 5.5 milliequivalents per liter (mEq./L.), while hypokalemia (hypopotassemia) exists when serum potassium levels are less than 3.5 mEq./L. This is not to say that serum potassium levels outside of the normal range must of necessity produce electrocardiographic or clinical manifestations. Moreover, identically abnormal serum potassium concentration in two persons need not be associated with the same degree of electrocardiographic change. In all probability other electrolytes—sodium in particular—modify the effect of abnormal potassium levels on the heart. Sodium and potassium ions have a somewhat antagonistic relationship, in that hyponatremia (low serum sodium) exaggerates the electrocardiographic effects of an elevated serum potassium level, while a normal or high serum sodium level tends to antagonize the effects of hyperkalemia. Undoubtedly, the mechanism responsible for the interrelationship of body potassium and sodium is far more complicated than the term "antagonism" would infer.

Etiology of Abnormalities of Serum and Body Potassium

Exogenous intake.—Excessive or deficient dietary intake of potassium is important to the extent that it aggravates the primary defect causing abnormal potassium retention or loss. The oral administration of potassium to subjects with normal renal function rarely leads to hyperkalemia, but potassium chloride given too rapidly or in too high concentrations by intravenous infusion can produce transiently lethal potassium levels (greater than 10 mEq./L.) despite excellent kidney function.

Renal function.—Chronically and severely diseased kidneys do not conserve potassium as well as do normal kidneys, and so, regardless of the intake of potas-

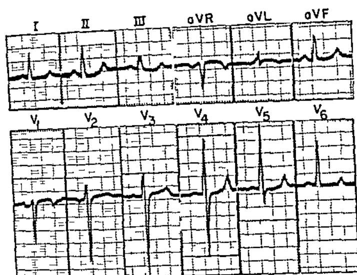
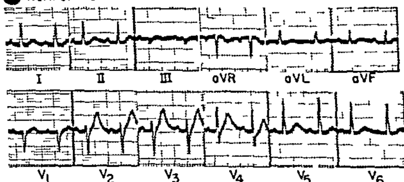


Fig 329—Electrocardiogram indicating hyperkalemia due to acute renal insufficiency The principal abnormality is the presence of upright tented T waves in leads I II aVF V V and V (The "tenting" of the T waves refers to their symmetrical and peaked contour) This finding occurs particularly when hyperkalemia is superimposed on pre-existing T wave abnormalities due to some other condition



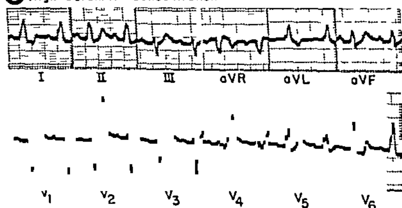
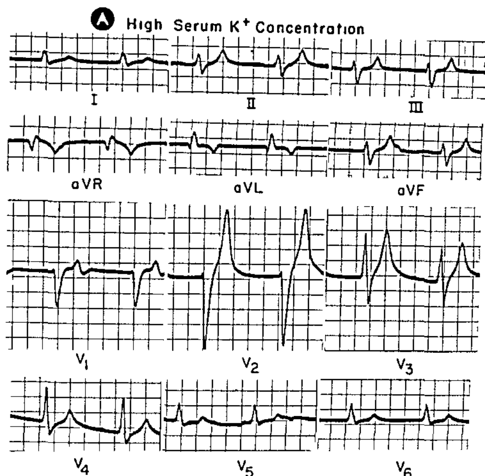


Fig 330—A electrocardiogram obtained shortly after onset of acute renal failure before any change in the serum potassium ion concentration This record is essentially within normal limits B record from same patient later in the course of acute renal failure when the serum potassium ion concentration was quite high The QRS interval is widened to 0 10–0 11 second and the QRS configuration resembles that seen in left bundle branch block The T waves are very tall in leads V through V and the S–T segments are depressed in leads I II aVF V and V In addition, there is slight prolongation of the P–R interval in the second electrocardiogram, as compared to the first These findings are compatible with hyperkalemia



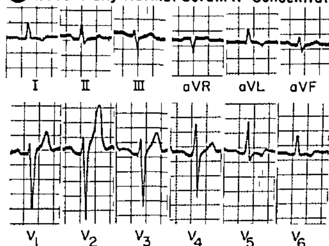
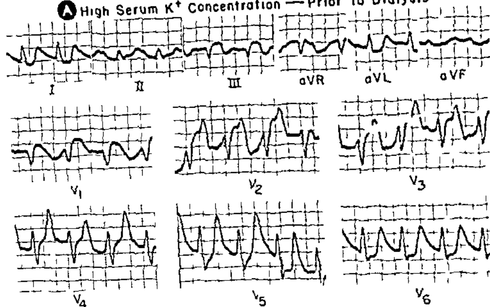
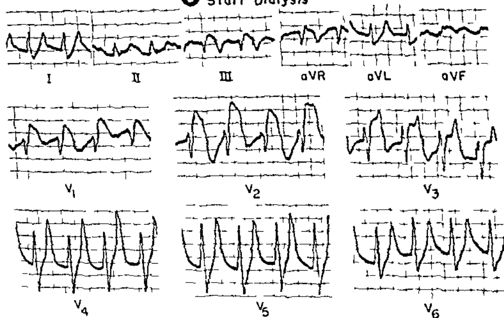


Fig 331—**A** electrocardiogram from a patient with severe renal failure and a markedly elevated serum potassium ion concentration This record shows a prolonged QRS interval (0 12 second) and tall peaked T waves in leads II III V, V, and V The P waves cannot be identified with certainty and so the mechanism of the rhythm may be atrioventricular nodal **B** later record from same patient The previous intraventricular conduction defect and the tall T waves have disappeared and the only abnormality present is T wave inversion in leads I V and V The QRS interval is 0 08 second Note that the general configuration of the QRS deflections is essentially the same in both records in corresponding leads despite the prolonged QRS interval in the former Thus in **A** there probably is a diffuse intraventricular block rather than a block in one or the other bundle branch The rhythm in **B** is sinus rhythm

sium the urinary concentration remains essentially constant According to Elkinton and his associates as well as other authorities the renal tubules continue to secrete potassium whether the serum potassium level is high normal or low This mechanism may be *chiefly responsible* for the low serum potassium concentrations often observed in patients with chronic renal disease Inasmuch as the potassium clearance in renal disease remains relatively constant the amount of potassium excreted by the kidneys is largely dependent on the volume of urine output *It is generally accepted* that either oliguria or anuria is necessary for the development of hyperkalemia However *the extent of the potassium intake protein catabolism* and potassium loss by other routes as well as the serum sodium concentration state of hydration and





through V The S–T segments are depressed and merge with terminal S waves in leads I aVL and V through V Also the T waves in most of the leads are larger than in corresponding leads in A. (*Continued*)

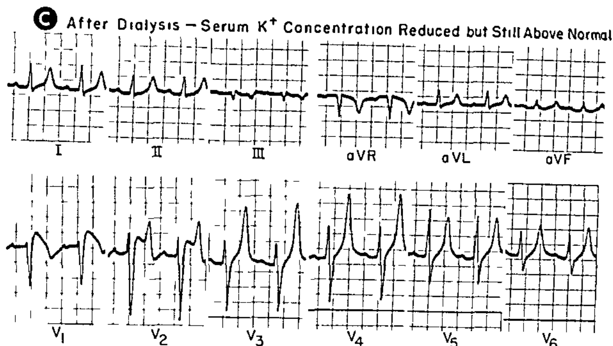


Fig 332 (*cont*) —**C** record after dialysis was completed at which time the serum potassium ion concentration was greatly reduced from its previous high level but still above normal The QRS interval in this electrocardiogram is 0 08 second The P–R interval is only slightly prolonged and the T waves are extremely tall and symmetrically peaked in leads I II and V through V_4 The S–T depression noted in **A** and **B** is much less striking in **C** but the S–T segment elevation in leads V_1 and V persists

other factors all influence the rapidity of onset and progression as well as the degree of hyperkalemia resulting from the primary renal dysfunction

Losses of potassium from the extracellular fluid — The common clinical examples of this mechanism which result in hypokalemia are excessive mercurial diuresis chlorothiazide therapy diabetic acidosis severe vomiting or diarrhea and the loss of gastro intestinal secretions through fistulas Untreated diabetic acidosis initially causes a rise in the serum potassium levels because of intracellular potassium depletion At a variable period after institution of therapy hypokalemia often appears probably owing to the following factors (*a*) excessive urinary loss of potassium secondary to the diuretic effect of glyco suria (*b*) dilution of the serum potassium by parenteral administration of large quantities of potassium free fluids and (*c*) the intracellular migration of potassium due to the acidosis per se and the intra cellular deposition of potassium and glycogen pro moted by insulin therapy

The ECG Features of Hyperkalemia

According to Merrill and his co workers the fol lowing general sequence of electrocardiographic changes develops as the serum potassium concentra tion becomes progressively more elevated

1 The T waves particularly in the precordial leads become tall narrow and peaked They may be of normal amplitude but tent shaped in the extremity and or the precordial leads in patients with a pre existing tendency to T wave inversion due to underlying cardiac disease
2 Prolongation of the Q–T interval may accompany the above finding
3 Then S–T segment depression occurs the S–T segment tending to form a more or less straight line from the nadir of the S wave to the peak of the T wave
4 Atrioventricular block may next appear usually in the form of a first degree atrioventricular block
5 There may be lowering and widening of the P waves with subsequent atrial standstill or atrial fibrillation
6 Intraventricular block of increasing degree is usu ally observed
7 Ventricular arrhythmia consisting of irregular bi zarre undulations often presages terminal ventric ular standstill
8 Ectopic ventricular rhythms such as extrasystoles tachycardia flutter and fibrillation may also be encountered in the advanced stages of hyper kalemia (Figs 329–334)

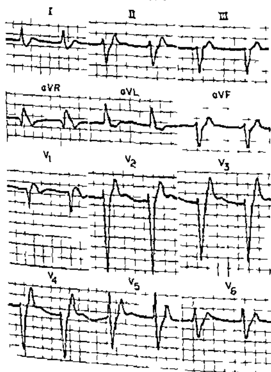
The ECG Features of Hypokalemia

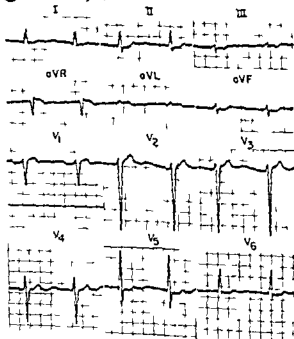
The electrocardiographic findings in hypokalemia

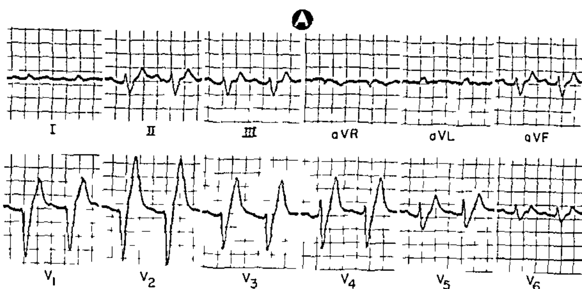
may reflect an intracellular deficit of potassium although Surawicz and Lepeschkin are of the opinion that the potassium gradient across the cell membranes *may* have a closer correlation with the changes in the electrocardiogram The abnormalities observed in hypokalemia are as follows

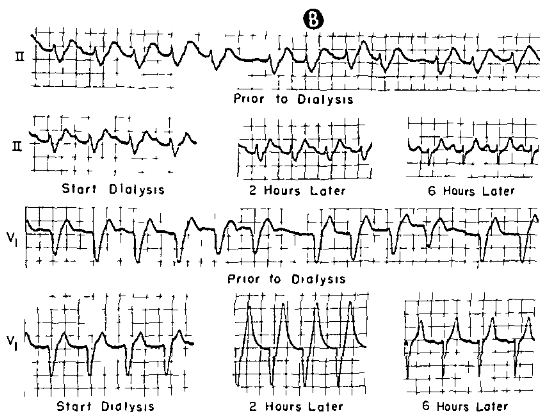
1 There is progressive lowering of the S–T segment *At first the ascending slope of the normal S–T segment* changes to a straight isoelectric line and then S–T segment depression of increasing degree appears as the serum potassium falls
2 There is lowering and finally inversion, of the T wave.
3 An increased amplitude of the U wave may be noted in the precordial leads
4 The Q–T interval is unchanged but often gives the appearance of being prolonged if the prominent U waves are confused for T waves
5 Weller and his associates have called attention to the increased *prominence of the P waves and* to the prolongation of the P–R interval which may often be seen in hypokalemia
6 Surawicz and Lepeschkin state that peaked P waves atrioventricular conduction disturbances and ectopic rhythms (particularly those of supraventricular origin) are helpful supportive evidence for hypokalemia when the electrocardiographic findings are otherwise equivocal (Figs 335 and 336)

Worthy of reiteration is the fact that *not uncommonly* the serum potassium concentration is found to be normal in the presence of an electrocardiogram typical of hyper or hypokalemia At the other extreme abnormally elevated or depressed serum potassium levels may not be attended by any detectable electrocardiographic changes









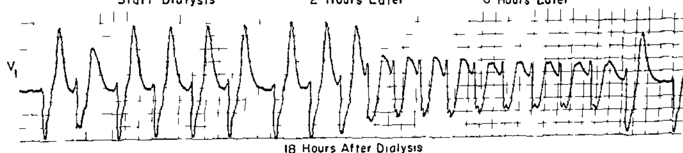
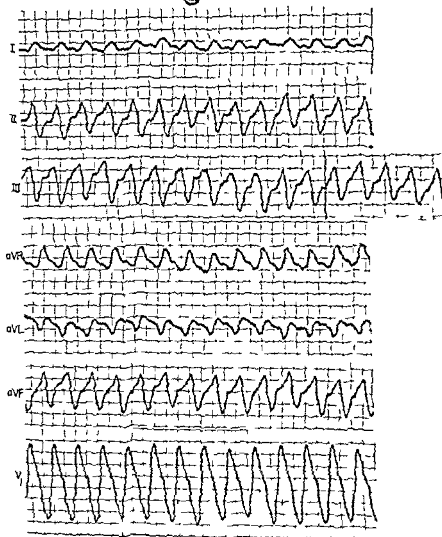
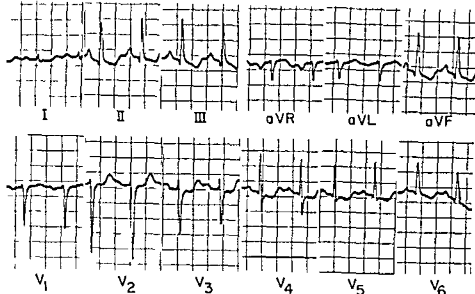
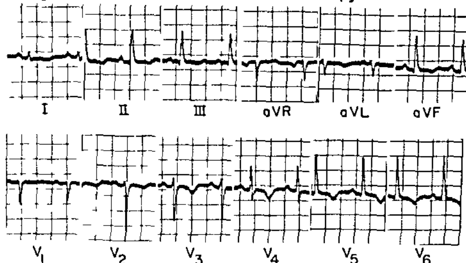


Fig 334 —A record from a patient in acute renal failure with greatly elevated serum potassium ion concentration The electrocardiographic abnormalities diagnostic of hyper prolonged intraventricular conduction and extremely tall T strips recorded before during and after dialysis with the arti



Terminal Record 36 Hours After Dialysis





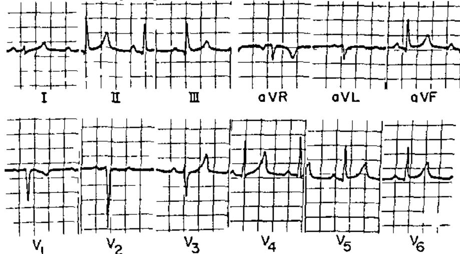


Fig 335 — **A** electrocardiogram from a patient with hypokalemia The changes related to the low serum potassium ion concentration are S–T segment depression in leads I II III and V_1 through V and inverted T waves in leads II III aVF V_4 and V_5 **B** record made about the time that parenteral potassium chloride therapy was begun This electrocardiogram is also diagnostic of hypokalemia but shows certain additional changes consisting of inversion of the T waves in leads I and V_2 through V The S–T segments are essentially isoelectric in most of the leads The patient was treated vigorously with potassium chloride and after therapy there was a moderately elevated serum potassium ion concentration **C** electrocardiogram consistent with moderately elevated serum potassium concentration showing tall peaked T waves in leads I II III aVF and V through V_6

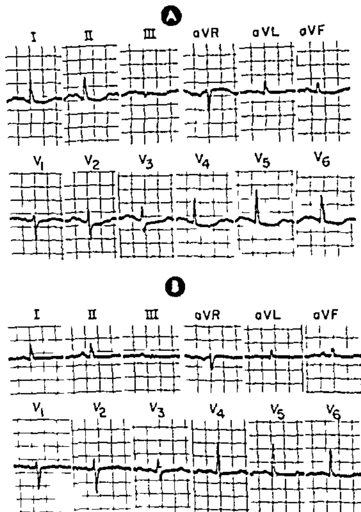


Fig 336 —A, typical electrocardiographic findings of hypokalemia, consisting of depressed S–T segments and shallowly inverted T waves in leads I II III aVF and V through V In addition prominent upright U waves can be seen shortly following the T waves in many of the leads The latter finding is frequently observed in hypokalemia and may sometimes lead to erroneous results in measuring the Q–T intervals in this condition B record made after the patient had been treated with potassium chloride and showing some improvement in the S–T segments and T waves compared with the first tracing However the T waves remain low in leads I, II aVF and V through V and the tracing on the whole is still suggestive of hypokalemia.

Calcium

Elevated serum calcium levels (hypercalcemia) rarely cause significant cardiac manifestations (other than shortening of the Q–T interval) unless they result from the intravenous infusion of calcium salts The latter route of calcium administration has been attended by occasional deaths particularly in patients with cardiac disease It is said that the danger of ventricular fibrillation resulting from elevated serum calcium levels is enhanced in digitalized patients for reasons unknown Transient ventricular ectopic rhythms have been described during intravenous infusion of calcium salts Abnormally low serum calcium levels (hypocalcemia) may cause Q–T interval prolongation (primarily by virtue of lengthening of the S–T segment) and produce lowering or inversion of the T waves

Ventricular Pre-Excitation (Wolff-Parkinson-White Syndrome)

THE Wolff Parkinson White syndrome is unique in that *it is one of the few clinical cardiac syndromes—if not the only one—in which the diagnosis rests entirely on the electrocardiographic findings* This condition usually has a benign prognosis and is characterized by the following features (*a*) in the electrocardiogram normal sinus P waves are followed after unusually short P–R intervals by QRS complexes of abnormal configuration and usually of prolonged duration and (*b*) patients with this condition are predisposed to recurrent episodes of paroxysmal ectopic rapid heart action

MECHANISM

It is generally conceded that pre excitation of a localized area of the ventricular musculature is responsible in the final analysis for the electrocardiographic features of this syndrome However the *mechanism by which pre excitation is accomplished* has not been established conclusively Two theories of the pre excitation mechanism—the accessory atrioventricular pathway theory and the accelerated atrioventricular conduction theory—will be considered in the following paragraphs

Accessory Atrioventricular Pathway Theory

The concept of accessory atrioventricular pathway proposes that one or more muscle bundles link atria to ventricles and serve as accessory conducting pathways competing more or less in this respect with the atrioventricular node (Such accessory pathways are presumed to be congenital in origin even though the clinical and electrocardiographic manifestations of *ventricular pre excitation may not appear for many years*) When the onset of pre excitation occurs it may be spontaneous or it may be the indirect result of disease of the normal conducting pathways *The anomalous conducting bundle provides a short cut* for the activation impulse enabling it to bypass the atrioventricular node which is normally the site of significant conduction delay

The pre excitation sequence may be summarized as follows

1 The atrial impulse is divided between the atrioventricular node and the accessory pathway However because of rapid conduction through the anomalous pathway the pre excitation impulse arrives in a focal region of the ventricular myocardium in advance *of the activation impulse traveling through the* atrioventricular junctional tissues Consequently the P–R interval is shortened

2 The depolarization wave may then pass from this one focus of pre excitation throughout both ventricles In this event the ventricular activation process is analogous to that in unifocal ventricular premature contractions in that depolarization spreads erratically from one ventricle to the other via the muscle fibers rather than along the conducting system Thus the QRS complex is abnormally widened and deformed

3 More often it happens that there is a synchronous activation of the ventricles by impulses passing *through both (a) the accessory pathway and (b)* the atrioventricular node and the bundle branch system This circumstance produces an electrocardio

graphic ventricular fusion beat the initial slurred portion of which (the delta wave) results from pre-excitation of one area of ventricular myocardium The rest of the QRS complex is produced largely by activation of remaining nonrefractory myocardium by the normally conducted impulse and may resemble somewhat the corresponding portion of a normally conducted beat in the same lead

4 It has been postulated that the length of the accessory atrioventricular pathway may influence the electrocardiographic features of the pre excitation beat. If the pathway is short the short P–R interval may be followed by a QRS complex of essentially normal configuration (for example the so called "syndrome of the short P–R interval normal QRS complex, and paroxysmal rapid heart action" which occurs predominantly in women) On the other hand a long anomalous conducting bundle may cause marked slurring of the initial portion of a widened and deformed QRS complex.

Accelerated Atrioventricular Conduction Theory

Prinzmetal and his associates present an alternative explanation for the pre excitation phenomenon based on their interpretation of certain clinical and experimental observations The fundamental tenets of their theory may be outlined as follows

1 Certain elements of the atrioventricular node and the bundle of His deliver the excitation impulse to specific regions of the ventricular myocardium

2 Pre excitation is the manifestation of accelerated conduction by some or all of the conducting elements in the atrioventricular junctional tissues

accelerated rate a ventricular fusion beat occurs as previously described

4 If all elements of the atrioventricular node evidence accelerated conduction the P–R interval is shortened just as previously but the QRS complex appears essentially normal since excitation spreads through the ventricles in the usual way

Validity of the Foregoing Theories of Ventricular Pre excitation

It is not feasible to enumerate in this review all of the evidence for and against each of the foregoing concepts of pre excitation Some of the more pertinent observations may be presented in condensed form as follows

Accessory atrioventricular pathway theory —1 Accessory muscle bundles have been identified in about one half of the cases with antemortem ventricular pre-excitation studied pathologically and have not been observed in normal human hearts (The frequent finding of conducting fibers between the atrioventricular node and the bundle of His or left bundle branch the "paraspecific bundles" of Mahaim has no pathologic relevance to the Wolff Parkinson White ... these conducting fibers are not to be

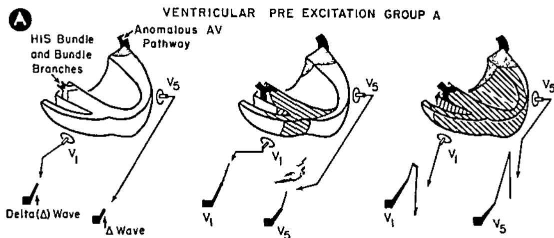
pulse directly into the ventricular myocardium by means of an artificial bypass or shunt analogous to the accessory muscle bundle

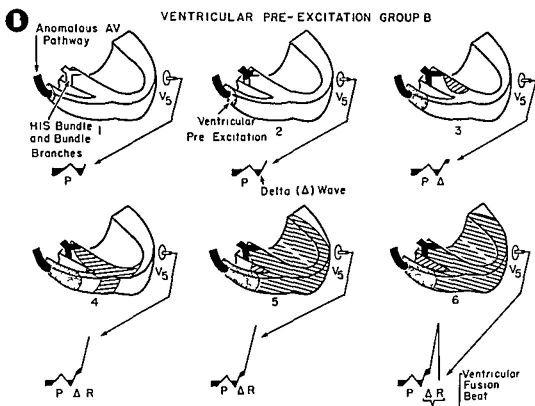
3 Atrioventricular node depressants such as carotid sinus pressure digitalis and parasympathomimetic drugs promote the accessory bundle as the preferential pathway for atrioventricular conduction The quantitatively different effects of these agents on normal and anomalous atrioventricular conduction seem to support albeit indirectly the existence of an alternate conduction pathway entirely separate from the normal atrioventricular conducting system

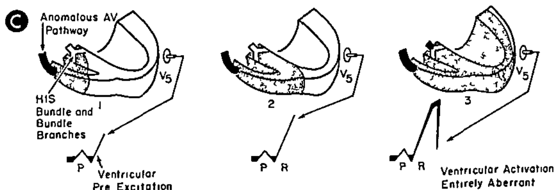
Accelerated atrioventricular conduction theory — 1 A point emphasized by proponents of this theory is that ventricular pre excitation beats have been produced by cardiac catheterization by traction on the pulmonary artery or the left auricular appendage by clinical and experimental coronary occlusion and

lieve that ventricular beats produced under the circumstances described do not represent pre excitation beats Thus Scherf has observed that stimulation of the interventricular septum elicits ectopic ventricular beats which appear at essentially the same rate as the rate of sinus discharge These beats may follow a short P–R interval and either may be totally distorted or may fuse with a normally conducted impulse to produce a ventricular fusion beat resembling that due to pre excitation According to other authorities atrioventricular nodal rhythms with atrioventricular dissociation and other arrhythmias apparently can produce complexes which may be confused with ventricular pre excitation beats

2 An observation which has been considered as favoring the accelerated atrioventricular conduction







Aberrant Ventricular Pre Excitation

Normal Ventricular Activation

Fig 337 —Legend on facing page

theory rather than the accessory pathway concept is that clinical and experimental atrioventricular block has been noted to abolish the pre-excitation phenomenon or to prevent its experimental production. However, ventricular pre-excitation beats have been observed clinically in cases of first and second degree atrioventricular block.

3. To some extent the validity of the accelerated atrioventricular conduction concept is indirectly compromised by the paradoxical effects of vagotonic and vagolytic drugs on the atrioventricular node and the pre-excitation mechanism. For example, if ventricular pre-excitation is due to abnormal conduction by a part of the atrioventricular node, then it must be assumed that the region of the atrioventricular node conducting the pre-excitation impulse and the remaining portion of the node respond differently to carotid sinus stimulation and the vagotonic cholinergic drugs, both of which tend to produce pre-excitation beats despite depressing nodal conductivity.

The authors of this text favor the accessory pathway concept of ventricular pre-excitation.

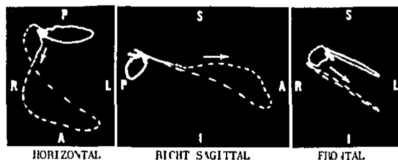
ELECTROCARDIOGRAPHIC FINDINGS

The characteristic features of ventricular pre-excitation and their incidences are cited below.

1. The P–R interval is 0.10 second or less in about 80% of the cases and is *rarely greater than 0.12 second*. A short P–R interval is significant provided the P waves are those of a normal sinoatrial rhythm rather than retrograde P waves.
2. The QRS interval tends to be greater than 0.10 second, measuring 0.11–0.12 second in almost one half of the cases.
3. The P–J interval* typically remains normal (not over 0.20 second), since the QRS deflection is lengthened at the expense of the P–R interval.

the major deflection is downward.

* The P–J interval extends from the onset of the P wave to the junction (J) of the QRS complex with the S–T segment.



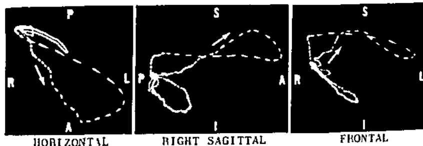
I II III aVR aVL aVF

V_1 V_2 V_3 V_4 V_5 V_6

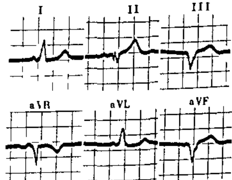
Fig 338 —ECG and VCG findings in Group A pattern of ventricular pre excitation

In the ECG the diagnostic abnormalities are P–R interval of 0 12 sec delta waves preceding upstrokes of R waves in leads II III aVF and V_1–V_6 downwardly directed delta wave in lead aVL and QRS interval of 0 12 sec Several findings—Q waves in leads I and V_6 and essentially normal T waves in all leads—are unusual in this pattern Generally the initial QRS forces in Groups A and B patterns are directed to the left and anteriorly so that Q waves are not usually recorded in transverse leads such as leads I and V

In the VCG the QRS sÊ loop is written almost entirely anteriorly at first slightly to the right and then to the left and inferiorly The anterior orientation of the QRS sÊ loop is responsible for the upright QRS deflections in all precordial leads of the ECG The initial and earliest portion of the QRS sÊ loop corresponds to the delta waves of the QRS deflections in the ECG and is written irregularly and abnormally slowly to the right anteriorly and inferiorly The closely spaced time markings in the initial and early portions of the QRS sÊ loop are pathognomonic of ventricular pre excitation The anterior location of the QRS sÊ loop is characteristic of the Group A pattern







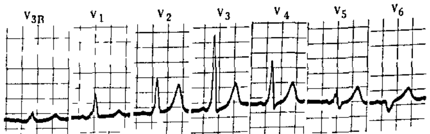


Fig 339 —Electrocardiographic and vectorcardiographic findings in Group A ventricular pre excitation

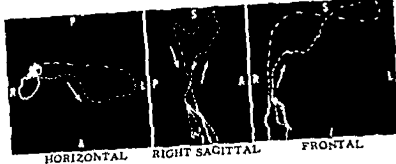
The electrocardiogram shows the short P–R interval prolonged QRS deflection and slurred delta wave characteristic of pre excitation while the upright QRS deflections in the right precordial leads identify the pattern type as Group A Note that leads III aVF and V display QS deflections a not infrequent finding in the Group A pre excitation pattern and one which is not indicative of myocardial infarction for reasons cited in the text

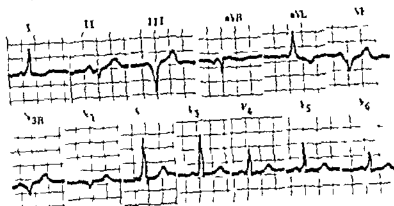
In the vectorcardiogram the QRS sÊ loop is written entirely anteriorly to the left and superiorly and displays the principal diagnostic feature of ventricular pre excitation — namely closely spaced time dashes in the initial and early portions of the efferent limb of the QRS sÊ loop Note that the appearance of the QRS sÊ loop as a whole is distorted

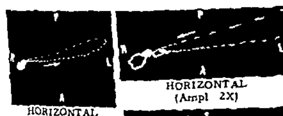
Fig 340 – Electrocardiographic and vectorcardiographic findings in the Group B ventricular pre-excitation pattern.

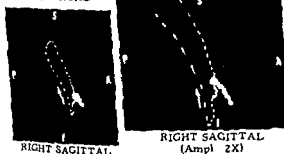
The electrocardiograms shows diagnostic features of ventricular pre-excitation The spatial angle between sÂ QRS and sÂ T is abnormally wide and represents a secondary T wave abnormality due to the altered ventricular activation process The downwardly directed QRS deflections in leads V and V and the upright QRS deflections in precordial leads to the left of V are typical of the Group B ventricular pre-excitation pattern

In the vectorcardiogram the QRS sÊ loop is written entirely superiorly and to the left and in the horizontal projection lies along the 0 axis, a finding typical of the Group B pattern. Note the slowed and irregular inscription of the efferent limb of the QRS sÊ loop The T sÊ loop is directed anteriorly and slightly to the right and for this reason, lead I records an inverted T wave.



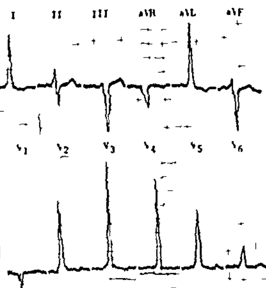


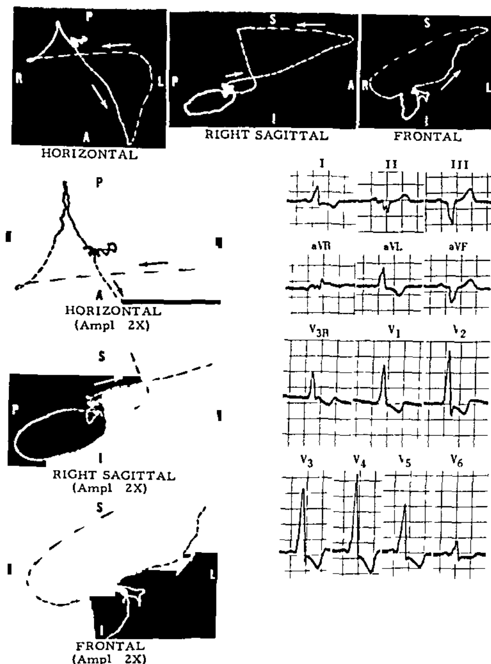




FRONTAL

FRONTAL (Ampl 2X)





72 with a history of recurring episodes of parox
the Group A ventricular pre excitation pattern

efferent limb of the loop displays closely spaced time ma
directed to the ... ntly posteriorly
sE loop in the
diographic and
ysmal tachycard
in Figure 343
observed in the

5 The configuration of the QRS deflection in the precordial leads divides the ventricular pre excitation syndrome into two groups as follows

In *Group A* the premature component and the remainder of the QRS complex are primarily upright in both left and right precordial leads In the left precordial leads *Q waves are absent* while the R waves *in right precordial leads are large* and often double peaked *The anterior direction of the activation forces has been interpreted as indicating that the accessory conducting bundle must be located posteriorly* (Fig 337 A)

In *Group B* the left precordial leads register the type of QRS complexes described above However leads V_1 and V show delta waves and QRS complexes which are resultantly negative QS deflections may be recorded in these right precordial leads as well as in leads II III and aVF The electrocardiographic pattern has been attributed to location of the accessory bundle anteriorly where it probably passes from the right atrium to insert on the epicardial surface of the right ventricle (Fig 337 B and C)

6 Secondary changes in the S-T segment and T wave may be observed in this syndrome the extent of the change depending on the QRS area Even though these changes may not be evident at times there is nonetheless instability of the ST-T complex Thus secondary S-T and T changes tend to appear for example with exercise or onset of sinus tachycardia This event needless to say does not indicate coronary insufficiency when aberrant atrioventricular conduction is known to exist

7 A spontaneous and variable shift back and forth between *normal* and *anomalous atrioventricular conduction takes place in about one half of the cases*

The features listed above apply to the recognition of the more typical cases of ventricular pre excitation but may also be of some aid in the identification of the less typical variants of this syndrome which have been catalogued by Ohnell (Figs 338–342)

PRODUCTION AND TERMINATION OF VENTRICULAR PRE EXCITATION

The effects of various agents on the pre excitation phenomenon in terms either of abolishing it if it is present or precipitating it in a susceptible person if it is not present are summarized in Table 31 The

TABLE 31 —The Effects of Carotid Sinus Stimulation and Various Drugs on Ventricular Pre excitation

Agent	Accessory Pathway	Atrioventricular Node	Result
Carotid sinus (vagal) stimulation	Insignificant effect	Increased vagal tone depresses conductivity	This procedure may produce pre-excitation beats or an atrioventricular nodal rhythm with normal QRS complex
Digitalis and parasympathomimetic drugs	Insignificant effect	Depress conductivity	Ventricular pre-excitation beats may result
Quinidine and pronestyl	Usually these drugs have a *greater depressant action on the* accessory pathway than on the atrioventricular node		In about 30% of cases quinidine regularly *terminates* ventricular pre-excitation
Sympathomimetic drugs	Apparently the accessory pathway *is not* able to conduct above a certain critical rate of drug (or exercise) induced sinus tachycardia	These drugs tend to enhance conductivity by neutralizing parasympathomimetic effects	Ventricular pre-excitation tends to be abolished
Atropine	Insignificant effects	This drug enhances atrioventricular node conductivity by virtue of its vagolytic action	Ventricular pre-excitation may be replaced by normal conduction through the atrioventricular node

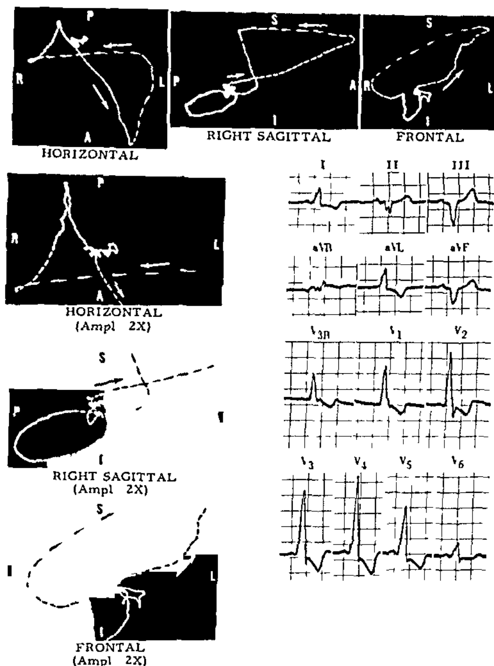


Fig 342 —Electrocardiogram and vectorcardiogram from a man 72 with a history of recurring episodes of parox ... characteristic of the Group A ventricular pre excitation pattern

directed to the ... posteriorly as evidenced by ... sE loop in the ... diographic and ... ysmal tachycar ... in Figure 343 ... observed in the

Group B

The following characteristics (see also Figs 340 341) are typical of this group

Horizontal QRS loop

In the vectorcardiographic pattern of Group B ventricular pre-excitation the initial deflection of the horizontal QRS loop ordinarily is written to the left and anteriorly but *occasionally is inscribed* to the right and anteriorly or posteriorly

The characteristic conduction delay and irregular inscription are noted in the early part of the QRS sE loop in each projection as was described in the Group A pattern

Generally the efferent limb of the horizontal QRS loop is written to the left and either slightly anteriorly or first anteriorly and then slightly posteriorly The loop then turns in a counterclockwise direction posteriorly and to the left and it remains in this quadrant until its inscription is completed

The maximal mean instantaneous vector of the horizontal QRS loop lies between −35 and +20

Right sagittal QRS loop

1 The orientation of the maximal mean instantaneous vector of the sagittal QRS loop ranges between +150 and −60

2 When the sagittal loop is oriented almost vertically superiorly as is often the case it usually has a counterclockwise direction of inscription If the loop is inferiorly located it usually is written in a clockwise direction Figure-of eight loops generally show counterclockwise inscription of their proximal components and clockwise *inscription of their* distal components

Frontal QRS loop

1 The maximal mean instantaneous vector of the frontal loop is generally situated between −45 and +30

2 A figure-of eight configuration of the frontal QRS loop is observed fairly commonly The proximal and distal components show counterclockwise and clockwise inscription respectively When the frontal QRS loop does not have this configuration it usually is written in a counterclockwise *direction*

S–T Vector and Ventricular Repolarization

Although in the series of patients with ventricular pre-excitation studied by us the number of patients was too small to attach any statistical significance to the data the findings may nevertheless represent the general trend and this is sufficient for the purposes of this text

Half of the vectorcardiograms both in Group A and in Group B showed displacement of the terminus *of the QRS sE loop indicative of an S–T vector* This vector was directed to the right in all cases in which it was present but its vertical and anteroposterior orientations varied widely

In Group A, the average orientations of the T sE loop in the horizontal right sagittal and frontal projections were +70 +100 and +75 respectively The corresponding values for T sE loop orientation in Group B were +130 +60 and +110 In two vectorcardiograms in Group B the horizontal T loop was either round or bifid but in either case the T loop showed two maximal instantaneous vectors of equal size but divergent duration In a third vector *cardiogram* the frontal T loop presented the features just described

PAROXYSMAL RAPID HEART ACTION IN THE WOLFF PARKINSON WHITE SYNDROME

Recurrent paroxysmal tachycardia accompanies this condition The paroxysmal tachycardias are almost invariably if not always of supraventricular origin Atrial and atrioventricular nodal tachycardias are most commonly noted followed in frequency by atrial fibrillation and atrial flutter (Figs 343–345) Although the occurrence of paroxysmal ventricular tachycardia in cases of Wolff Parkinson White syndrome has been reported in the past Wolff and other investigators believe that most or all of the apparent ventricular tachycardias observed in uncomplicated cases of the Wolff Parkinson White syndrome are actually supraventricular tachycardias with persistent anomalous atrioventricular conduction (Fig 344) Supportive evidence for this belief continues to accumulate

Mechanism

The mechanisms proposed in explanation of the rapid abnormal heart rhythms occurring in the Wolff

importance of these factors in the diagnosis and study of the Wolff Parkinson White syndrome is obvious Moreover Wolff and Richman emphasize that ventricular pre excitation produces QRS complexes which may mimic myocardial infarction and conversely that it also may obscure the presence of myocardial infarction In either case the most reliable diagnostic technique is to terminate the anomalous atrioventricular conduction long enough to evaluate the configuration of the QRS complexes in the absence of pre excitation Similarly this procedure permits the diagnostic features of associated cardiac abnormalities like right ventricular hypertrophy and right bundle branch block to appear

VECTORCARDIOGRAPHIC FINDINGS

The vectorcardiographic features to be described below were observed by the authors of this text in 14 patients having the Wolff Parkinson White syndrome whose electrocardiograms and ancillary clinical data were quite typical of this condition In 6 of these patients the QRS configuration in the precordial electrocardiographic leads was that of the Group A type of ventricular aberration while the remaining 8 patients could be placed in the Group B type By and large the vectorcardiograms in these two groups were in keeping with the abnormalities present in the precordial electrocardiograms so that the vectorcardiograms like the electrocardiograms could be divided into two general pattern types which for consistency will be given the same designations as the corresponding electrocardiographic patterns The salient feature diagnostic of ventricular pre excitation and common to both types of QRS sÊ loop configuration was the fact that the initial and early portions of the spatial QRS loop were inscribed slowly erratically and in an abnormal direction This abnormality which corresponds to the delta wave or slurred initial limb of the QRS deflection of the electrocardiogram is rarely if ever observed in the absence of ventricular pre excitation A more detailed description of the QRS sÊ loop in Groups A and B ventricular pre excitation follows

Group A

This entity presents the following characteristics (see also Figs 338 and 339)

Horizontal QRS loop

1 The initial defection of the horizontal QRS loop is written to the left and usually anteriorly although occasionally posteriorly
2 In all three projections of the QRS sÊ loop the earliest portion of the loop and sometimes almost the entire efferent limb shows conduction delay and an irregular or erratic inscription
3 In this type of QRS sÊ loop configuration the efferent limb proceeds to the left and relatively far anteriorly and then the loop turns in a counterclockwise direction the afferent limb passing behind the efferent limb to return on the right and anteriorly Occasionally the entire horizontal QRS loop is inscribed in a clockwise direction and lies anteriorly and to the left
4 The maximal mean instantaneous vector of the horizontal QRS loop ranges in orientation between +20° and +95

Right sagittal QRS loop

1 The QRS sÊ loop in the sagittal projection usually has a figure of eight configuration the proximal and distal components of the loop being inscribed in counterclockwise and clockwise directions respectively
2 The initial deflection of the sagittal QRS loop is directed anteriorly and inferiorly in most instances
3 The sagittal QRS loop is almost invariably situated anteriorly and either slightly superiorly or inferiorly In our cases the maximal mean instantaneous vector of the sagittal loop ranged in orientation between −30 and +70

Frontal QRS loop

1 The frontal QRS loop tends to vary widely in configuration although figure of eight loops were relatively common in the cases which we studied In figure of eight frontal loops the proximal component of the loop usually is written in a counterclockwise direction and the distal portion of the loop in a clockwise direction When the efferent and afferent limbs of the frontal loop do not cross each other the direction of inscription ordinarily is counterclockwise Occasionally the frontal QRS loop has an almost linear configuration
2 The maximal mean instantaneous vector (or long axis) of the frontal QRS loop usually is located between −30° and +40

Group B

The following characteristics (see also Figs 340 and 341) are typical of this group

Horizontal QRS loop

1 In the vectorcardiographic pattern of Group B ventricular pre-excitation the initial deflection of the horizontal QRS loop ordinarily is written to the left and anteriorly but occasionally is inscribed to the right and anteriorly or posteriorly
2. The characteristic conduction delay and irregular inscription are noted in the early part of the QRS sÊ loop in each projection as was described in the Group A pattern
3 Generally the efferent limb of the horizontal QRS loop is written to the left and either slightly anteriorly or first anteriorly and then slightly posteriorly The loop then turns in a counterclockwise direction posteriorly and to the left and it remains in this quadrant until its inscription is completed
4 The maximal mean instantaneous vector of the horizontal QRS loop lies between −35 and + 20

Right sagittal QRS loop

1 The orientation of the maximal mean instantaneous vector of the sagittal QRS loop ranges between +150 and −60
2. When the sagittal loop is oriented almost vertically superiorly as is often the case it usually has a counterclockwise direction of inscription If the loop is inferiorly located it usually is written in a clockwise direction Figure-of-eight loops generally show counterclockwise inscription of their proximal components and clockwise inscription of their distal components

Frontal QRS loop

1 The maximal mean instantaneous vector of the frontal loop is generally situated between −45 and +30
2 A figure-of eight configuration of the frontal QRS loop is observed fairly commonly The proximal and distal components show counterclockwise and clockwise inscription respectively When the frontal QRS loop does not have this configuration it usually is written in a counterclockwise direction

S-T Vector and Ventricular Repolarization

Although in the series of patients with ventricular pre-excitation studied by us the number of patients was too small to attach any statistical significance to the data the findings may nevertheless represent the general trend and this is sufficient for the purposes of this text

Half of the vectorcardiograms both in Group A and in Group B showed displacement of the terminus of the QRS sÊ loop indicative of an S-T vector This vector was directed to the right in all cases in which it was present but its vertical and anteroposterior orientations varied widely

In Group A the average orientations of the T sÊ loop in the horizontal right sagittal and frontal projections were +70 +100 and +75 respectively The corresponding values for T sÊ loop orientation in Group B were +130 +60 and +110 In two vectorcardiograms in Group B the horizontal T loop was either round or bifid but in either case the T loop showed two maximal instantaneous vectors of equal size but divergent duration In a third vectorcardiogram the frontal T loop presented the features just described

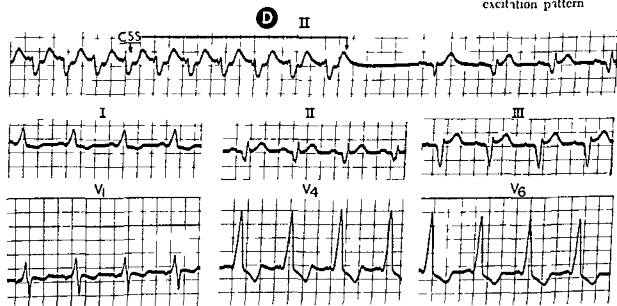
PAROXYSMAL RAPID HEART ACTION IN THE WOLFF-PARKINSON-WHITE SYNDROME

Recurrent paroxysmal tachycardia accompanies such a high percentage of cases of Wolff Parkinson White syndrome (at least 70%) that it has come to be recognized as one of the principal clinical features of this condition The paroxysmal tachycardias are almost invariably if not always of supraventricular origin Atrial and atrioventricular nodal tachycardias are most commonly noted followed in frequency by atrial fibrillation and atrial flutter (Figs 343–345) Although the occurrence of paroxysmal ventricular tachycardia in cases of Wolff Parkinson White syndrome has been reported in the past Wolff and other investigators believe that most or all of the apparent ventricular tachycardias observed in uncomplicated cases of the Wolff Parkinson White syndrome are actually supraventricular tachycardias with persistent anomalous atrioventricular conduction (Fig 344) Supportive evidence for this belief continues to accumulate

Mechanism

The mechanisms proposed in explanation of the rapid abnormal heart rhythms occurring in the Wolff

Fig 343 —Electrocardiographic lead strips recorded from the patient with *Wolff Parkinson White* syndrome whose vectorcardiogram was shown in Figure 342 Leads I II III V_1 V and V in column **A** were recorded before quinidine therapy The leads in **B** were recorded after the patient had received quinidine 0 2 Gm at 2 hour intervals for 5 doses Note the prolongation of both P–R and QRS intervals Also note that while the QRS configuration in leads I II and III has not changed significantly in **B** as compared to **A** the precordial leads in **B** display QRS deflections characteristic of the Group *B* pre excitation pattern whereas in **A** the Group A pre-excitation pattern was present The lead strips in **C** were recorded shortly after those in **B** and demonstrate a paroxysmal tachycardia of supraventricular origin with a ventricular rate of 130 beats per minute The Group B pre excitation pattern persists despite onset of the tachycardia In **D** the lead strip of lead II was recorded during carotid sinus stimulation (*CSS*) During the period of carotid sinus stimulation there is gradual slowing of the rate of the ectopic tachycardia until it stops abruptly at the point indicated by the second arrow fol [illegible] III V_1 V and V were obtained shortly after the paroxysmal tachycardia was terminated and demonstrate return to the Group A pre excitation pattern



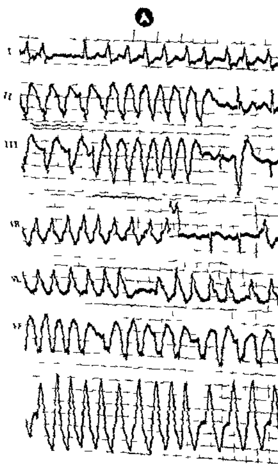
Parkinson-White syndrome will be described in the paragraphs to follow

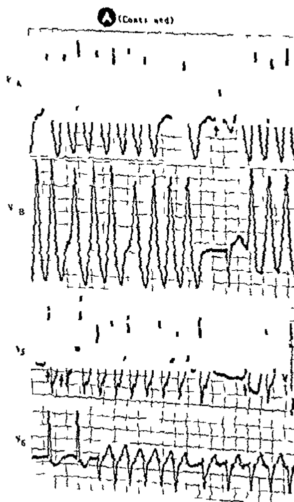
Paroxysmal atrial and atrioventricular nodal tachycardias—A paroxysm of rapid heart action may be precipitated by passage of the atrial or atrioventricular nodal activation impulse downward through the atrioventricular node and its subsequent return to the atria or atrioventricular node via the accessory pathway thus constituting a form of re-entry. If the re-entry is repetitive the paroxysmal tachycardia becomes established. Consistent with this explanation is the fact that the last ventricular beat preceding onset of an atrial or nodal tachycardia and also the ventricular complexes written during the tachycardia are usually not of the pre-excitation type

Paroxysmal atrial fibrillation and flutter—Unlike the preceding tachycardias atrial fibrillation and flutter cannot be explained satisfactorily on the basis solely of echo re-entry of the impulse (into the atria) via the accessory pathway since impossibly high ventricular rates would have to be assumed. However some investigators postulate that the mechanism initiating the paroxysm may be re-entry but that the impulse is returned to the atria very early. If it arrives during the partial refractory or "vulnerable" period of atria the re-entry impulse may cause onset of atrial

Fig 344—Electrocardiogram and vectorcardiogram from patient hospitalized with clinical picture of acute con

pre-excitation (Continued)





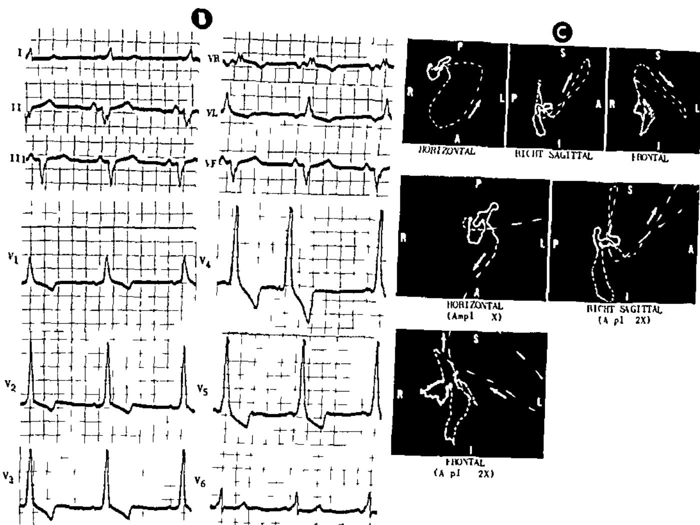


Fig 344 (*cont*) —The ventricular pre excitation to the left and superiorly the efferent limb of the Q ...

While a number of cases of ventricular tachycardia occurring in ventricular pre excitation (*Wolff Parkinson White* syndrome) have been reported in the past most authorities feel at present that many if not all of these cases were actually atrial fibrillation with the pre excitation type of aberration of the ventricular complexes

fibrillation or flutter (During these arrhythmias a rapid almost constant stream of atrial impulses passes down either the accessory bundle or the atrioventricular node and bundle branch system depending on which is less refractory at the time of onset of the tachycardia Since one of these two conducting pathways continues to be used exclusively the ventricular beats if they are of the pre excitation type usually are greatly widened and deformed If ventricular pre excitation beats with marked ventricular aberration should occur during atrial fibrillation the electrocardiogram may resemble ventricular tachycardia quite closely The differentiation of the two rhythms assumes critical importance in this instance since the patient may be treated unnecessarily with potentially hazardous drugs such as quinidine in the belief that ventricular tachycardia is present Actually the rapid rhythm should be treated like any other atrial fibrillation with a rapid ventricular rate—that is, with digitalis A paroxysm of pre excitation beats occurring in *atrial fibrillation* will usually terminate when there is a ventricular pause of sufficient length to permit recovery of atrioventricular nodal conductivity During the interval of transition from anomalous to *normal atrioventricular* conduction ventricular fusion beats of the type observed commonly in ventricular *pre excitation* with sinus rhythm may appear and they are soon followed by the appearance of QRS deflections of normal configuration *and duration*

Ventricular tachycardia —See comments concerning this type of tachycardia in *the preceding three* paragraphs

CLINICAL ASPECTS OF WOLFF-PARKINSON-WHITE SYNDROME

White syndrome would be

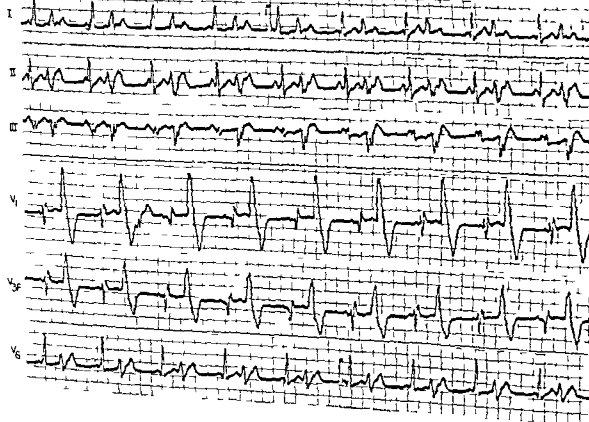
of ventricular pre-excitation is frequently erroneously as being indicative of heart disease (*b*) Ventricular pre-excitation may obscure other abnormalities in the electrocardiogram (*c*) The Wolff-Parkinson-White ... accompanied by a high incidence ...

bigem

lar pre
point
pre-exc
not infrequently
White variety

over a period of time may produce irreversible changes in normal hearts or seriously compromise diseased hearts

However in cases of the Wolff-Parkinson-White syndrome ill effects have been encountered so infrequently that the benign nature of this condition is justifiably stressed. On the other hand according to life insurance experience the mortality rate among applicants with Wolff-Parkinson-White syndrome is about three times normal



PART V

Illustrative Vectorcardiograms and Electrocardiograms

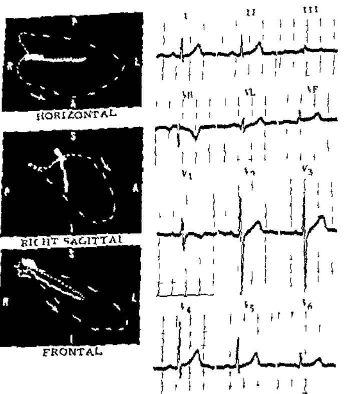
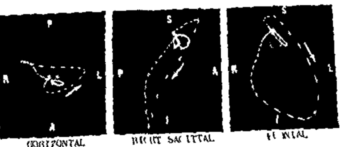


Fig 346 —Electrocardiogram and vectorcardiogram recorded from a normal girl 13

In the electrocardiogram the R/S amplitude ratio of 1 in lead V is not abnormal in this age group or in younger children

In the vectorcardiogram the corresponding finding is the anterior orientation of the first half of the QRS sE loop Note that a perpendicular through the midpoint of lead V would bisect the horizontal QRS loop almost equally the first half lying on the positive side of lead V and the second half on the negative side of lead V hence the equiphasic RS deflection in lead V Also note the right posterior terminal deflection of the horizontal QRS loop this too is a normal vectorcardiographic finding in this age group



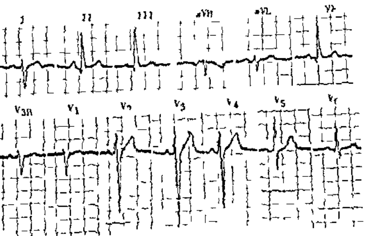


Fig 347 —Electrocardiogram and vectorcardiogram in a normal boy 5

In the electrocardiogram the slight slurred S of A QRS and the terminal S waves in I and V and V are normal findings in this age group

In the vectorcardiogram the related features are horizontal QRS loop showing a prominent rightward and posterior terminal deflection and frontal QRS loop oriented almost vertically *inferior* the first half of the loop being written to the left and the second half to the right of the midline

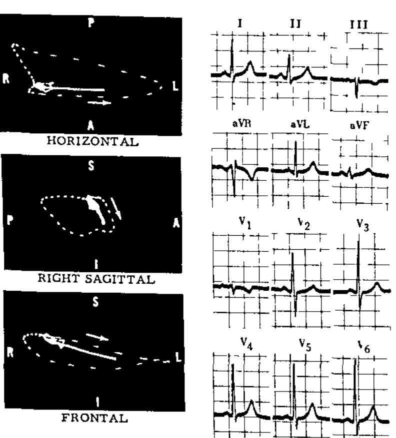


Fig 348 —Electrocardiogram and vectorcardiogram in a normal woman 24 Note the right posterior terminal deflection of the horizontal QRS loop of the vectorcardiogram An unusual feature of the vectorcardiogram is that the frontal QRS loop although directed almost horizontally to the left is written in a clockwise direction Usually in frontal loops with this orientation the direction of inscription is counterclockwise

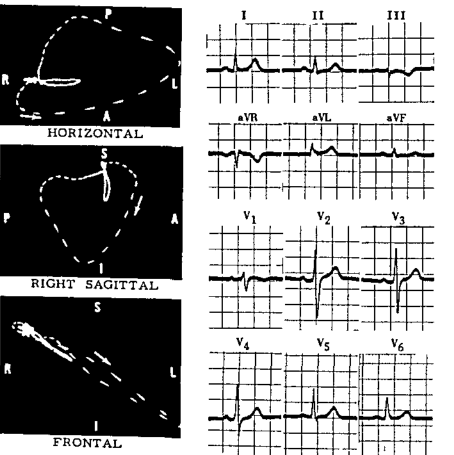


Fig 349 —Electrocardiogram and vectorcardiogram in a normal man 50 Note that the long axes of the planar QRS loops and planar T loops are directed almost parallel although the spatial angle subtended by the long axes of the QRS sÊ and T sÊ loops can occasionally be as wide as 45 in normal persons

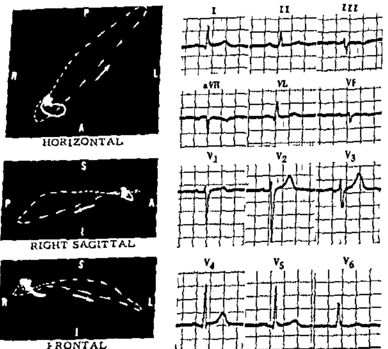


Fig 350 —*Normal electrocardiogram* and abnormal vectorcardiogram in man 58 with angina pectoris

The electrocardiogram shows no definite S–T segment or T wave abnormalitie. The exact width of the horizontal QRS–T angle cannot *be determined precisely* because none of six precordial leads record a transitional T wave and therefore the orientation of Â T can only *be approximated*. All that can be stated concerning the orientation of Â T in the horizontal plane is that the vector lies between +30 and +90 while *Â QRS is situated at* about −20

In the vectorcardiogram the spatial angle subtended *by the long axes* of the QRS sÊ and T sÊ loops is 65. This angle is abnormally wide since the spatial angle of 45 represents the upper *limits of normal in* vectorcardiograms recorded with the cube lead system

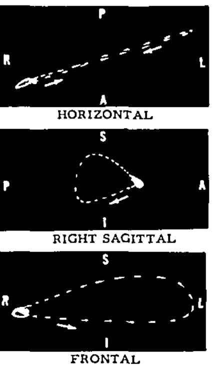
HORIZONTAL
RIGHT SAGITTAL
FRONTAL

HORIZONTAL (Ampl 2X)
RIGHT SAGITTAL (Ampl 2X)
FRONTAL (Ampl 2X)

I II III
aVR aVL aVF
V_1 V_2 V_3
V_4 V_5 V_6

Fig 351 —Electrocardiographic and vectorcardiographic pattern of left ventricular hypertrophy. Note in the vectorcardiogram the posterior rotation of the horizontal QRS loop, the left axis deviation of the frontal QRS loop and the discordant T sÊ loop

Fig 352 —Electrocardiographic and vectorcardiographic patterns of left ventricular hypertrophy Note the leftward posterior and superior orientation of the QRS sÊ loop and the discordant T sÊ loop



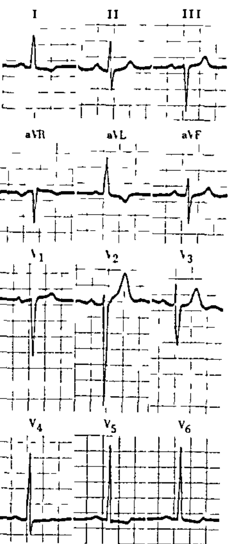


Fig 353 —Electrocardiographic and vectorcardiographic patterns of left ventricular hypertrophy The above vectorcardiogram was recorded with much less amplification than the normal vectorcardiograms appearing in this text Thus the mean instantaneous vectors of the QRS sÊ loop are of much greater magnitude than normal and this is reflected in the electrocardiogram by the deep S waves appearing in leads V_1 and V and the tall R waves in leads V through V The T sÊ loop is discordant to the QRS sÊ loop in the vectorcardiogram

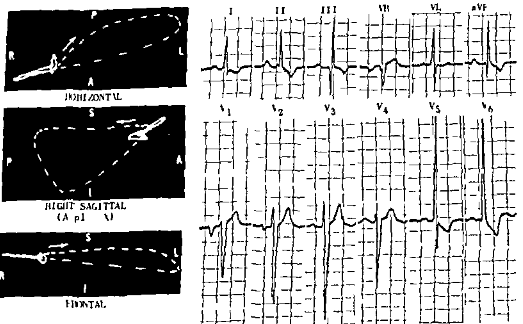


Fig. 354.—Electrocardiographic and vectorcardiographic pattern of left ventricular hypertrophy in a youth 15 with congenital aortic stenosis. Note in the electrocardiogram the deep Q waves in lead III and aVF. This finding is related in the vectorcardiogram to the clockwise inscription of the frontal QRS loop which causes the early portion of the loop to be written superiorly. The diminished R wave amplitude in lead V of the electrocardiogram is explained by the clockwise direction of inscription and posterior displacement of the efferent limb of the horizontal QRS loop of the vectorcardiogram. The electrocardiogram is otherwise quite typical of left ventricular hypertrophy. The explanation for the *reversed direction of inscription of the QRS sÊ loop in its three planar projections* is not known but this finding has been observed by the authors of this text and by other investigators in occasional vectorcardiograms of patients with left ventricular hypertrophy. The vectorcardiogram otherwise is quite consistent with left ventricular hypertrophy.

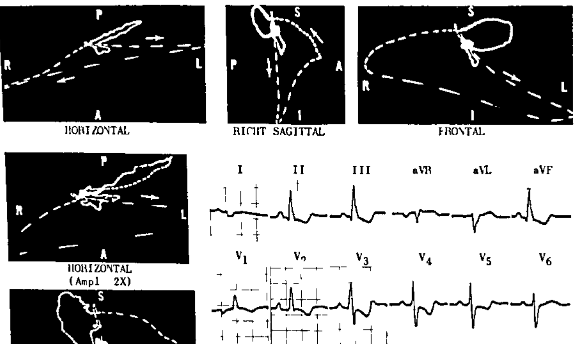


Fig 355 (*left*) —Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy in a woman 48 with interatrial septal defect

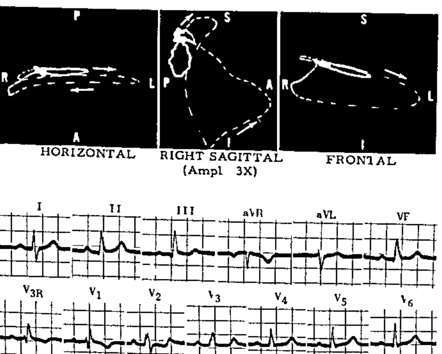


Fig 356 (*right*) —Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy in a woman 33 with interatrial septal defect Note the RSR deflections in leads V_{R} and V in the electrocardiogram In the past the electrocardiographic diagnosis of incomplete right bundle branch block would have been made on the basis of the QRS configuration in the right precordial leads In the vectorcardiogram the horizontal QRS loop is mainly clockwise inscribed and is located anterior The sagittal QRS loop has a figure of eight configuration the larger distal portion of the loop being counterclockwise inscribed These features are consistent with the vectorcardiographic diagnosis of right ventricular hypertrophy The authors of this text interpret the above electrocardiogram as being consistent with right ventricular hypertrophy Their diagnostic criteria with particular reference to the RSR deflection in lead V are as follows (1) the R amplitude exceeds the amplitude of the initial R wave and (2) the R /S amplitude ratio is equal to or greater than 1





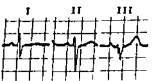


Fig 357 —El ctroc rdi graphic and vect rc rdi gr phic pat rn of right ventricul r hypertrophy in a wom n 30 with tetralogy f Fallot




Fig 358 —El ctroc rdiogram and vectorc rdiogram in ventricular septal defect The RSR defl ctions in lead V and V of the electrocardiogram are suggestive of right ventricular hypertrophy while the very tall R waves in leads V and V are equally suggestive of left ventricul r hypertrophy The horizontal QRS loop of the vectorcardiogram i inscribed in a clockwise direction and presents the type of configuration desig nated in the text as the RSR pattern of right ventricular hypertrophy However the efferent limb of the horizontal QRS loop is written farther to the left and somewhat more posteriorly than is usually the c se in this QRS loop pattern of right ventricular hypertrophy The counterclockwise direction of inscription of the right sagittal QRS loop is con i tent with right ventricular hypertrophy but the countercl ckwise inscrip tion and relatively horizontal orientation of the frontal QRS loop suggest coexi ting left ventricular hypertrophy The authors of this text interpret both the el ctroc rdiogram and vectorcardiogram as being suggestive of combined ventricular hypertrophy

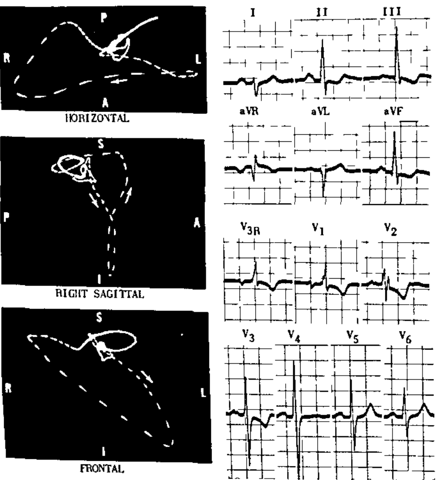


Fig 359 —Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy *in a patient with mitral stenosis*

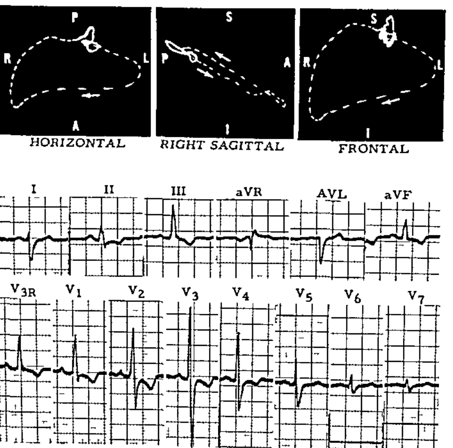
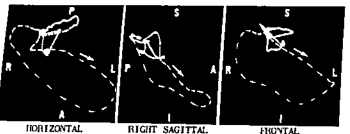


Fig 360 —Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy in a patient with mitral stenosis



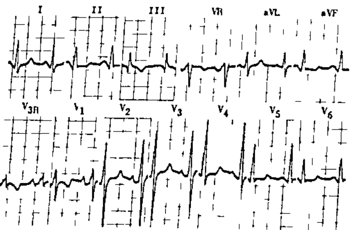
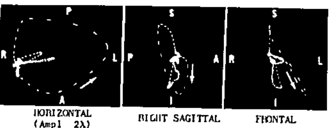


Fig 361 —Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy in a patient with mitral stenosis

In the electrocardiogram lead V and V₁ display wide initial R waves the R/S amplitude ratio in lead V being greater than 1 These are the only electrocardiographic features suggestive of right ventricular hypertrophy

In the vectorcardiogram however the horizontal QRS loop is written entirely in a clockwise direction anteriorly a finding which is diagnostic of right ventricular hypertrophy



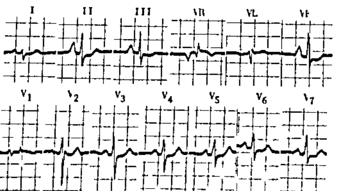


Fig 362 —Electrocardiogram and vectorcardiogram in mitral stenosis

In the electrocardiogram the tall P wave in leads II III and aVF are suggestive of right atrial enlargement while the right axis deviation of Â QRS and the small but upright QRS deflection in lead V₁ are suggestive of right ventricular hypertrophy

In the vectorcardiogram however the P sÊ loop is oriented for the most part posteriorly and is otherwise consistent with the diagnosis of left atrial enlargement The posterior and superior terminal deflection of the QRS sÊ loop and the inscription of the efferent limb of the QRS sÊ loop farther anteriorly than is usually the case in the normal vectorcardiogram are vectorcardiographic findings commonly seen in patients with mitral stenosis These QRS sÊ loop findings cannot be considered indicative per se of right ventricular hypertrophy although there is evidence suggesting that they may be related to enlargement of the pulmonary outflow tract

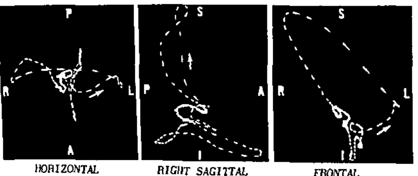


Fig 363 —Electrocardiogram and vectorcardiogram in a woman 69 with mitral stenosis pulmonary hypertension and congestive heart failure

In the electrocardiogram the P waves are 0 12 second in duration and are notched or slurred—findings suggestive of the P mitrale pattern of left atrial enlargement The marked right axis deviation of A QRS and the QR deflections in leads V_{3R} and V_1 are compatible with right ventricular hypertrophy While the resultantly positive QRS voltage in lead I small as it is might be thought to indicate marked left axis deviation of Â QRS the more likely the possibility is that there is marked right axis deviation of Â QRS and that the latter presents in the electrocardiogram as left axis deviation because of the downward slant of the effective axis of lead I in a left to right direction (See Chapter 4 for detailed discussion)

In the vectorcardiogram the P sÊ loop slows large instantaneous vectors directed both anteriorly and posteriorly and so combined atrial hypertrophy and enlargement is probably present The planar QRS loops present the features of the type B QRS sÊ loop pattern observed in some patients with mitral stenosis or chronic cor pulmonale Note the marked right axis deviation of the frontal QRS loop The above vectorcardiogram is interpreted by the authors of this text as being indicative of right ventricular hypertrophy

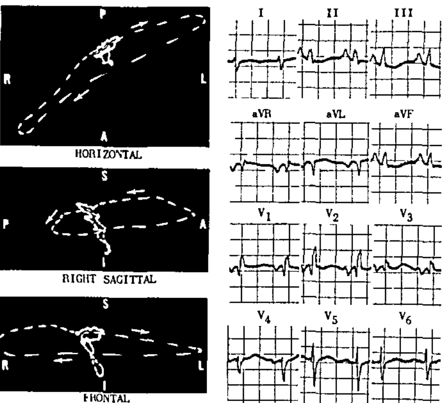


Fig 364 —Electrocardiographic and vectorcardiographic patterns of right ventricular hypertrophy in a patient with chronic cor pulmonale

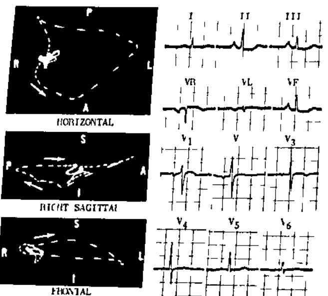


Fig 365 —Electrocardiogram and vectorcardiogram in chronic pulmonary emphysema without evidence of chronic cor pulmonale.
In the electrocardiogram the S–T segment depression in leads II, III and aVF and the low T waves in lead I and V through V are probably related to digitalis effect. Otherwise the record is noncontributory with reference to the diagnosis of chronic pulmonary emphysema.
The vectorcardiogram shows the type A QRS sÊ loop pattern observed in patients with mitral stenosis or with chronic pulmonary emphysema without cor pulmonale and an S–T vector compatible with digitalis effect.

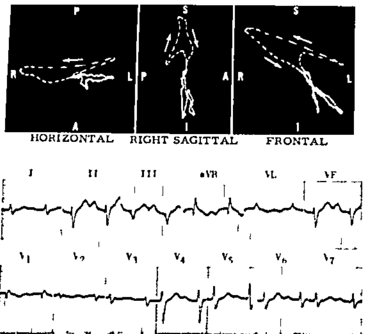


Fig 366 —Electrocardiogram and vectorcardiogram in chronic cor pulmonale.
The electrocardiogram shows first-degree atrioventricular block, the S_I–S_{II}–S_{III} pattern in the limb leads, QR deflections in lead V_1, and the precordial lead pattern of "clockwise rotation," findings compatible with chronic cor pulmonale.
The vectorcardiogram displays the type A QRS sÊ loop pattern, but in contrast with the vectorcardiogram in Figure 365 here the large terminal instantaneous vectors of the QRS sÊ loop directed to the right posteriorly and superiorly are strongly suggestive of right ventricular hypertrophy. The anterior and vertically inferior orientation of the P sÊ loop and increased magnitude of the maximal mean instantaneous vector of the P sÊ loop are consistent with right atrial enlargement.

Fig 367 —Electrocardiographic and vectorcardiographic patterns of right atrial enlargement and right ventricular hypertrophy in a patient with chronic cor pulmonale The type B QRS sÊ loop pattern in the vectorcardiogram is considered by the authors of this text to be diagnostic of right ventricular hypertrophy Note the large anteriorly oriented P sÊ loop

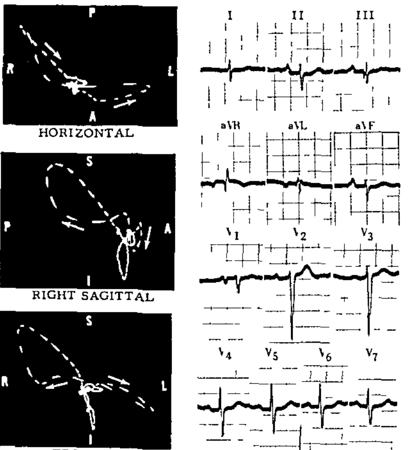
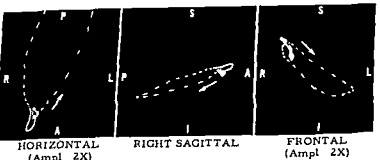


Fig 368 —Electrocardiogram and vectorcardiogram recorded in chronic cor pulmonale The electrocardiogram shows no evidence suggesting the presence of right ventricular hypertrophy while the vectorcardiogram displays the type B QRS sÊ loop pattern considered by the authors of this text to be indicative of right ventricular hypertrophy The large P sÊ loop directed somewhat anteriorly and vertically inferiorly is consistent with the vectorcardiographic diagnosis of right atrial enlargement



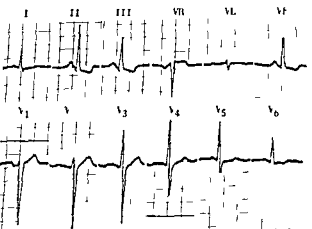
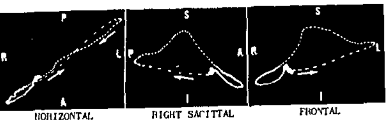


Fig 369 —The vectorcardiographic type C QRS sÊ loop pattern in chronic pulmonary emphysema. The S–T segment and T wave abnormalities in the electrocardiogram and the S–T vector and the T sÊ loop abnormalities in the vectorcardiogram are related—in part at least—to digitalis effect.



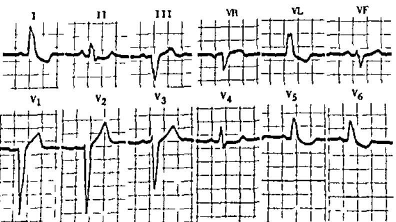
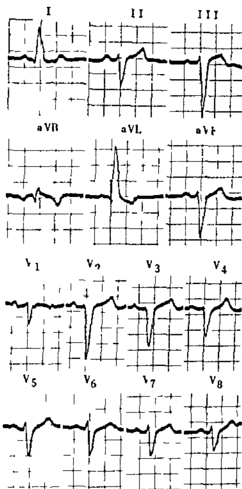


Fig 370 —Electrocardiogram and vectorcardiogram in left bundle branch block. In the electrocardiogram the QRS duration is 0.11 second. In the vectorcardiogram the QRS sÊ loop is written initially slightly anteriorly and to the left and there is marked conduction delay in the entire efferent limb of the loop. The horizontal QRS loop has a figure-of-eight configuration.





wise rotat ow

waves being recorded by all six chest leads There is marked left axis deviation of Â QRS in the frontal plane

In the vectorcardiogram the QRS sÊ loop is written rly and then eriorly Ter posteriorly and superiorly the afferent limb of the loop showing conduction delay

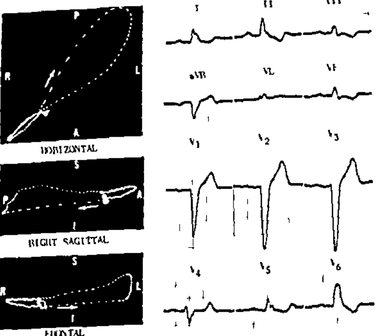


Fig 372 —Electrocardiogram and vectorcardiogram in left bundle branch block

In the electrocardiogram the QRS duration is 0.14 second

In the vectorcardiogram the QRS sE loop is written immediately to the left posteriorly and inferiorly and there is conduction delay in the entire afferent limb of the loop



Fig 373 —Electrocardiogram and vectorcardiogram in a variant type of left intraventricular block (left ventricular hypertrophy with terminal conduction delay)

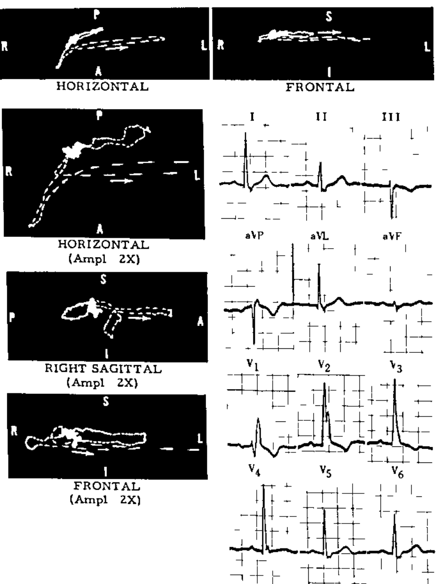


Fig 374 —Electrocardiogram and vectorcardiogram in the common type of right bundle branch block

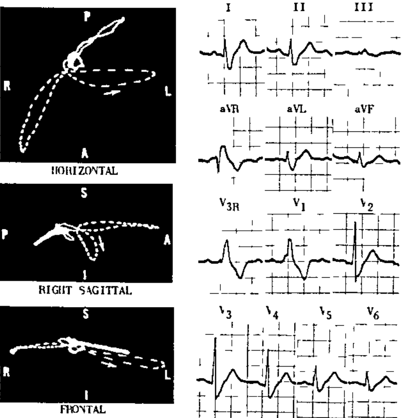


Fig 375 —Electrocardiogram and vectorcardiogram in the common type of right bundle branch block

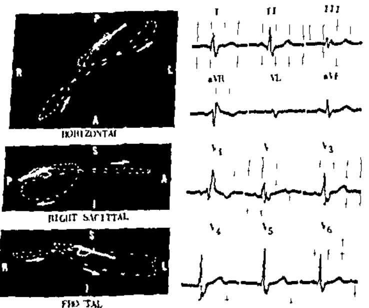


Fig 376 —Electrocardiogram and vectorcardiogram in the common type of right bundle branch block

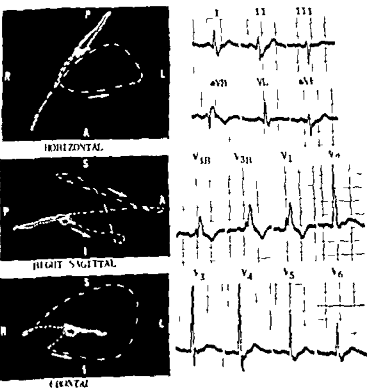


Fig 377 —Electrocardiogram and vectorcardiogram in the variant type of right bundle branch block

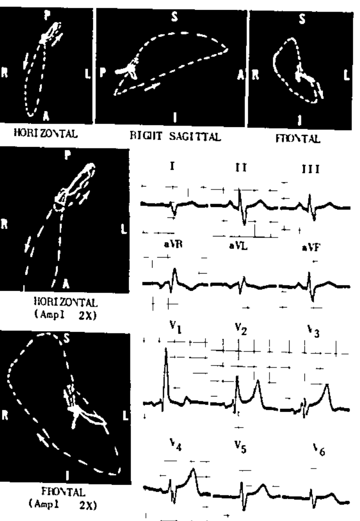


Fig 378 —Electrocardiogram and vectorcardiogram in the variant type of right bundle branch block

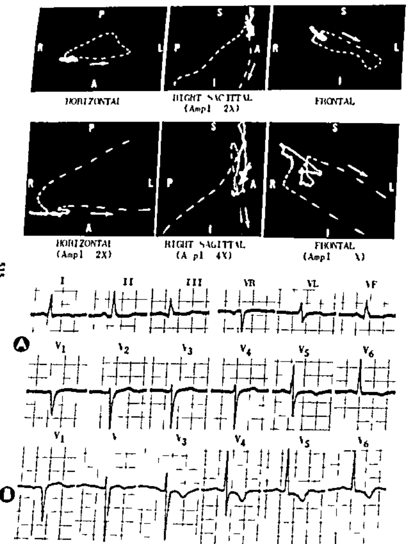


Fig 379 —Electroc rdiogram and vectorc rdiogr m from a wom n C8 with a history of myoc rdi l inf rction 1 y r previously

Th tw lve l ad el ctrocardiogram in **A** w s obt ined t the same time a the vectorc rdiogr m hown above wh le the six precordi l lead in **B** were recorded 2 months later In **A** there are inverted T w ves in le d I V and V Le d V di plays a QS deflecti n and there are low initi l R waves of incr sing amplitude in l ad V through V None of these findings constitute suffici nt evidence to ju tify the diagno s of antero pt l myocardi l infarcti n The precordi l lead in **B** show somewh t t ller R waves in le d V V and V and more m rked T wave inversi n in le d V through V

In th vectorc rdiogr m th QRS sÊ loop i writt n initially to the l ft po teriorly and superiorly There is an interior conc vity in th e rly portion of the efferent limb of th QRS sÊ lo p in th h riz nt l and right sagitt l proj ction The initi l porti n of th right s gittal QRS loop is written in a dir ction th r ver of norm l Th e QRS sÊ lo p finding are diagno tic of antero pt l myoc rdi l infarcti n Th T sÊ loop i di cord nt to th QRS sÊ loop and there i n S-T vector directed pred min ntly to the rig t nd superiorly Th e finding are c nsi tent with anterol ter l subendoc rdi l myoc rdial inj ry and i ch mi The vectorc rdiogr m is th refore diagno tic of ol ant rosept l myocardi l infarcti n and subend c rdi l injury and ischemia

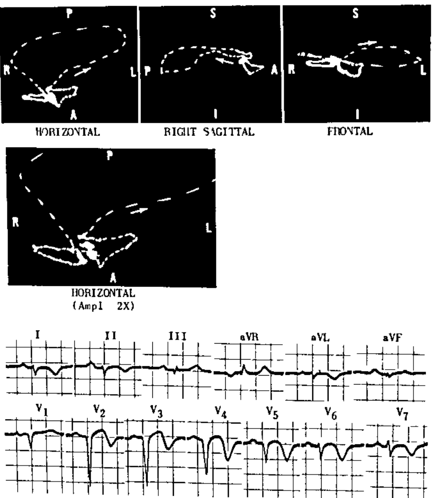


Fig 380 —Electrocardiogram and vectorcardiogram in a patient with recent anteroseptal myocardial infarction

Note in the electrocardiogram the QS deflections in leads V_1 through V_6 and the inverted T waves in these leads and in leads I II aVL V_6 and V_7. The terminal S waves in leads I II aVL V_6 and V may reflect the type of peri infarction block described by Grant and associates in which the terminal instantaneous QRS vectors are directed away from the initial instantaneous vectors of the QRS sÊ loop

In the vectorcardiogram note that the QRS sÊ loop is written immediately to the left posteriorly and superiorly and that there is an anterior concavity in the outgoing limbs of the horizontal and sagittal loops An S–T vector is directed to the right and anteriorly and the T sÊ loop is directed to the right and slightly posteriorly These findings are compatible with anteroseptal myocardial infarction and subepicardial myocardial injury

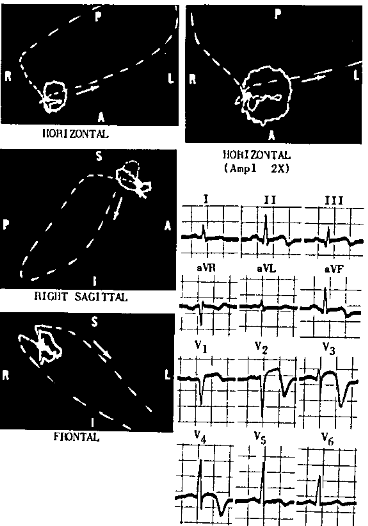


Fig 381 —Electrocardiogram and vectorcardiogram in acute anterior myocardial infarction

In the electrocardiogram note the elevated S–T segments in leads V_1 through V and the deeply inverted T waves in leads V through V_4. Leads V_1 and V_2 display QS deflections and lead V displays a small Q wave preceding a low R wave

In the vectorcardiogram the QRS sÊ loop is written initially to the right anteriorly and superiorly and then is inscribed to the left posteriorly and inferiorly This produces an anterior concavity in the early portion of the efferent limb of the QRS sÊ loop in its horizontal and sagittal projections and reverses the direction of inscription of the initial deflection of the sagittal QRS loop A small S–T vector is directed almost straight anteriorly in the horizontal projection The T sÊ loop is rounded in the horizontal projection and written with an almost uniform rate of inscription to the left and posteri

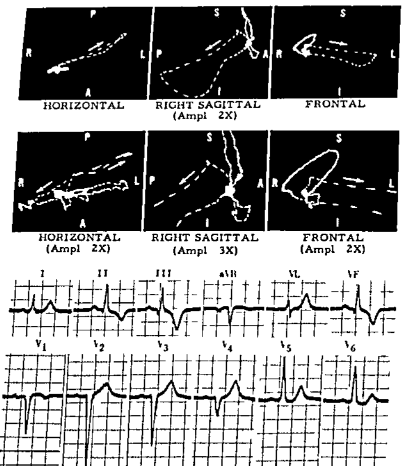


Fig 382.—Electrocardiogram and vectorcardiogram in old healed anteroseptal myocardial infarction.

Note in the electrocardiogram that leads II, III and aVF display deeply inverted T waves while leads V through V show low R waves and lead V a QS deflection. These findings are interpreted as diaphragmatic myocardial ischemia and old anteroseptal myocardial infarction.

In the vectorcardiogram the horizontal QRS loop is written initially to the left and posteriorly and shows posterior displacement of almost the entire efferent limb of the loop. In the sagittal projection the posterior displacement of the efferent limb causes a complete reversal in the direction of inscription of the sagittal loop. The TsÊ loop is directed to the left, slightly posteriorly and markedly upwards, and there is an S-T vector directed to the right. Thus the vectorcardiogram is in agreement with the findings in the electrocardiogram.

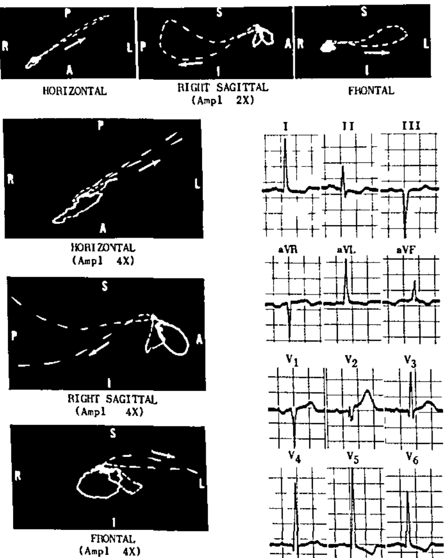


Fig 383 —Electrocardiogram and vectorcardiogram in old strictly anterior myocardial infarction with coexisting left ventricular hypertrophy

In the electrocardiogram tall R waves and inverted T waves are recorded in leads I aVL V_5 and V_6 These findings are consistent with the diagnosis of left ventricular hypertrophy while the small Q wave preceding the R wave in lead V and the small Q wave in lead V_3 are suggestive of old strictly anterior myocardial infarction

In the vectorcardiogram the horizontal QRS loop is written initially to the right and anteriorly and then the loop turns in a clockwise direction and is written to the left and posteriorly There is an anterior concavity in the early portion of the efferent limb of the horizontal QRS loop and the long axis of the loop is displaced posteriorly The right sagittal QRS loop is written briefly anteriorly although not easily seen and then is written almost directly posteriorly There is left axis deviation of the frontal QRS loop the mean instantaneous vectors of the planar QRS loops are of greater than normal magnitude and the T sÊ loop is discordant to the QRS sÊ loop The latter vectorcardiographic findings are diagnostic of left ventricular hypertrophy while the abnormalities initially described are diagnostic of old strictly anterior myocardial infarction

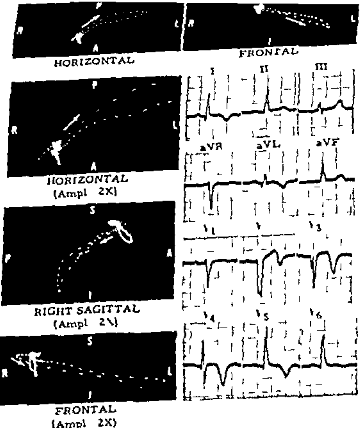


Fig 384 —Electrocardiogram and vectorcardiogram in recent anterior myocardial infarction

In the electrocardiogram lead V records a low initial R wave and an inverted T wave while lead V displays a QS deflection and a deeply inverted T wave. Inverted T waves also appear in leads I aVL, V and V. These findings are consistent with the diagnosis of anterior myocardial infarction and are suggestive of recent as opposed to old healed infarction

In the vectorcardiogram the horizontal QRS loop is written initially to the right and anteriorly and then the loop is inscribed in a clockwise direction posteriorly and to the left. There is posterior displacement of the efferent limbs of the horizontal and right sagittal QRS loops. The T sE loop is directed anteriorly slightly to the left and inferiorly. It should be pointed out that the vectorcardiogram was recorded several days after the electrocardiogram and so the T sE loop orientation is not consistent with the T wave abnormalities observed in the electrocardiogram

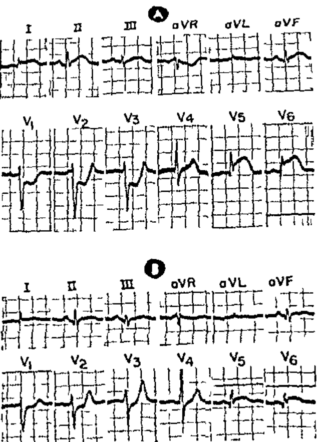


Fig 385 —Acute anterolateral myocardial infarction. These two electrocardiograms were recorded 24 hours apart from the same patient. In A the most prominent findings consist of marked S–T segment elevation in leads I II aVF V and V and marked S–T depression in leads V_1 through V. While small Q waves are present in lead I V and V they are not sufficiently deep or wide to constitute reliable evidence of anterolateral infarction. However the subepicardial injury pattern present in the left lateral leads is strongly suggestive of an early stage of developing anterolateral infarction. In B the S–T segments are returning to the isoelectric level and the Q waves are relatively more prominent

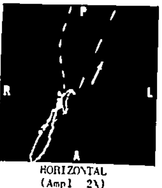
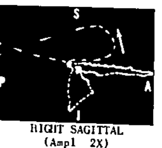
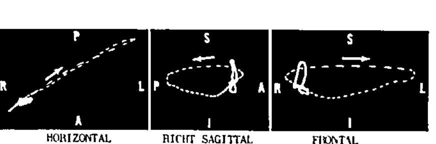




Fig 386 —Electrocardiogram and vectorcardiogram in recent diaphragmatic or inferior myocardial infarction In the vectorcardiogram note the superior displacement of the efferent limb of the QRS sÊ loop and the superior orientation of the T sÊ loop in the right sagittal and frontal projections As was the case in Figure 384 here too the fact that the vectorcardiogram was recorded several days after the electrocardiogram accounts for the more prominent abnormalities diagnostic of diaphragmatic infarction in the vectorcardiogram as compared to the electrocardiogram



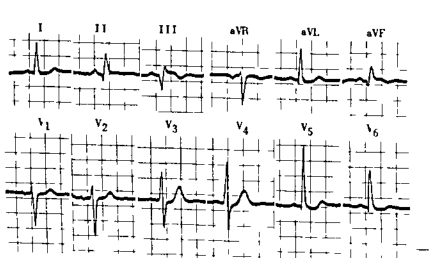


Fig 387 —Electrocardiogram and vectorcardiogram in old healed diaphragmatic or inferior myocardial infarction and left ventricular hypertrophy

In the electrocardiogram lead II and aVF record abnormal Q waves but there is a small initial R wave in lead III These findings suggest old diaphragmatic lateral myocardial infarction The tall R wave in lead V is consistent with the diagnosis of left ventricular hypertrophy as is the T wave inversion in leads I V₄ and V

In the vectorcardiogram the findings are QRS sÊ loop rotated abnormally posteriorly and superiorly rightward and superior displacement of early portion of efferent limb of frontal QRS loop and an inferior concavity in remaining portion of this limb The inscription of the first portion of the right sagittal QRS loop is reversed in direction and in both frontal and right sagittal projections the mean 0 02 second instantaneous QRS vector lies superior to −40 The T sÊ loop is discordant to the QRS sÊ loop These findings are diagnostic of old diaphragmatic lateral myocardial infarction and coexisting left ventricular hypert...

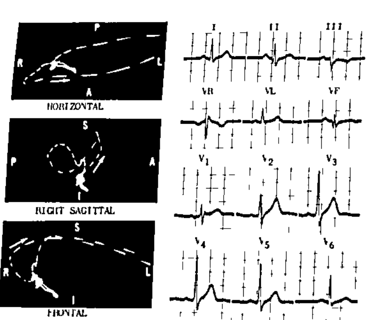


Fig 388 —Electrocardiogram and vectorcardiogram in old or healed diaphragmatic myocardial infarction Note that in the electrocardiogram lead II records an initial Q wave while lead III and aVF register small initial R waves followed by S waves The small initial R waves in lead III and aVF are produced by the brief initial deflection of the QRS sE loop directed inferiorly while the S waves in the preceding leads reflect the rightward and/or superior displacement of the efferent limb of the QRS sE loop Thus the S waves preceded by small R waves in lead III and aVF are to be considered the equivalent of abnormal Q waves The electrocardiographic and vectorcardiographic findings just described are consistent with the diagnosis of old healed diaphragmatic myocardial infarction

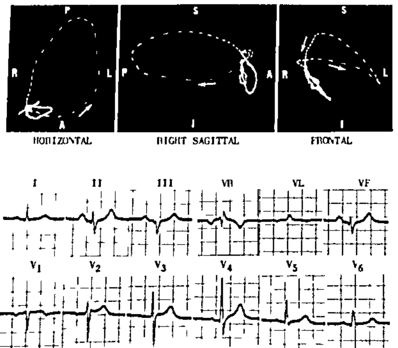


Fig 389 —Electrocardiogram and vectorcardiogram in old diaphragmatic myocardial infarction Note in the electrocardiogram that lead II and aVF register small Q waves while lead III records a QS deflection The corresponding finding in the vectorcardiogram is the superior displacement of the efferent and afferent limbs of the QRS sE loop The significance of the posterior rotation of the QRS sE loop and the anterior concavity in the early portion of the efferent limb of the horizontal QRS loop is not apparent Both the electrocardiographic and the vectorcardiographic findings are consistent with the diagnosis of old diaphragmatic infarction

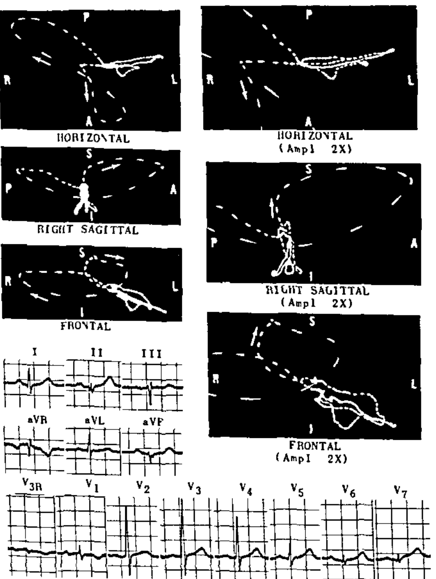


Fig 390—Electrocardiogram and vectorcardiogram in old healed diaphragmatic posterolateral myocardial infarction

The electrocardiogram displays a small Q wave in lead II while leads III and aVF register small initial R waves. In leads V R and V the *R/S amplitude ratio is greater than 1 while leads V and* V register very low R waves preceded by small Q waves. These findings are somewhat suggestive of old diaphragmatic posterolateral infarction

In the vectorcardiogram however the first half of the QRS sĒ loop is displaced abnormally far anteriorly to the right and superiorly while the terminal portion of the QRS sĒ loop is written far to the right and posteriorly. The mean 0.02 second instantaneous QRS spatial vector calculated from the QRS sĒ loop lies well to the right and abnormally anteriorly and superiorly. The QRS sĒ loop abnormalities are quite diagnostic of diaphragmatic posterolateral infarction without evidence of recent onset of the infarction

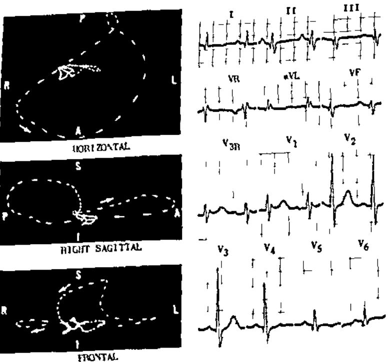


Fig 391 —Electrocardiogram and vectorcardiogram in old posterolateral myocardial infarction

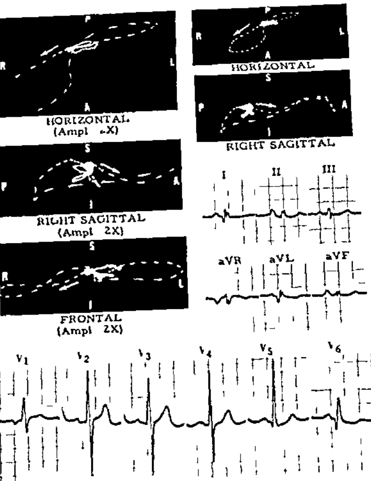


Fig 392 —Electrocardiogram and vectorcardiogram in old postero lateral myocardial infarction Note the relatively tall and wide R wave in I and V which is the counterpart of the deep wide Q waves in leads I and V

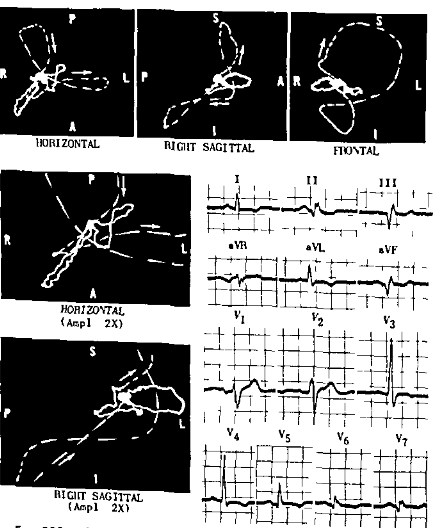


Fig 393 —Electrocardiogram and vectorcardiogram in old diaphragmatic posterolateral infarction Note the abnormal Q waves in leads II III aVF V_6 and V in the electrocardiogram In the *vectorcardiogram* the initial portion of the QRS sÊ loop is written abnormally far to the right anteriorly and superiorly while there is anterior and medial displacement of the *afferent* limb of the loop The T sÊ loop is directed to the right and anteriorly These *vectorcardiographic findings* are consistent with the diagnosis of diaphragmatic posterolateral infarction probably old with posterolateral myocardial ischemia

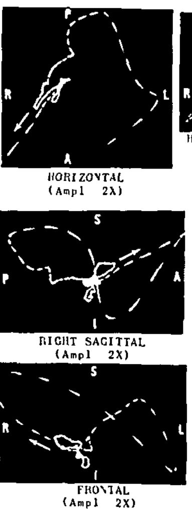
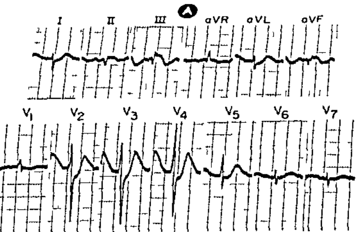


Fig 394 —Electrocardiogram and vectorcardiogram in old healed diaphragmatic posterolateral myocardial infarction



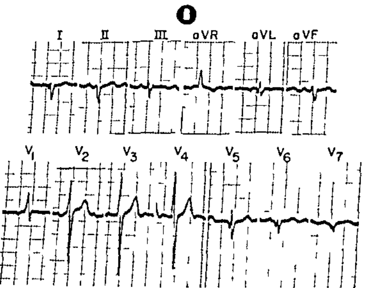


Fig. 395.—Evolution of electrocardiographic changes in acute posterolateral infarction. Electrocardiograms **A** and **B** were recorded about 1 week apart.

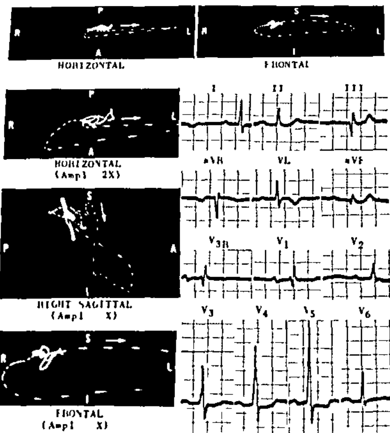


Fig. 396.—Electrocardiogram and vectorcardiogram in old strictly posterior myocardial infarction. Note the RSR′ deflections in leads V and V in the electrocardiogram, the conventional interpretation of which would be "incomplete right bundle branch block." In the vectorcardiogram, however, there is marked anterior displacement of the efferent limb of the QRS sE loop consistent with the diagnosis of old strictly posterior infarction. (The patient had been hospitalized with the clinical diagnosis of myocardial infarction 1 year prior to these recordings.)

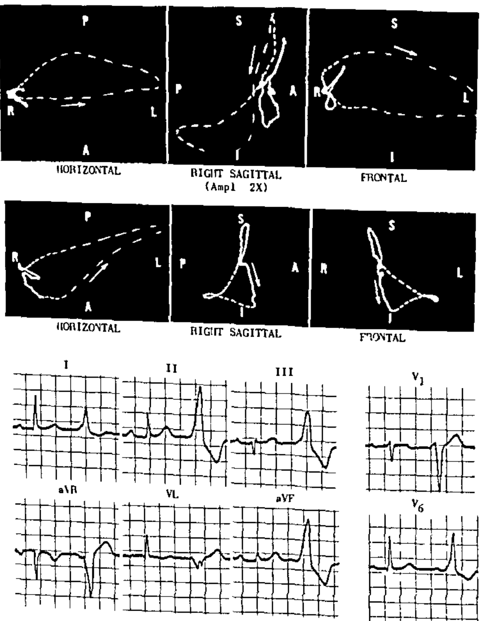


Fig 397 —Electrocardiogram and vectorcardiogram in old diaphragmatic myocardial infarction

In the electrocardiogram only leads V_1 and V of the precordial leads are shown in addition to the six routine extremity leads Note that lead III displays an RSR deflection while lead aVI records in RR deflection In each of the limb leads the conducted sinus beat is followed by a coupled ventricular extrasystole

The vectorcardiogram shown in the top row corresponds to the conducted sinus beats in the electrocardiogram Note the large superiorly directed early deflection of the right sagittal QRS loop and the superior displacement of the efferent limb of the frontal *QRS loop* These findings plus the superior orientation of the T sÊ loop are compatible with diaphragmatic *myocardial* infarction and diaphragmatic ischemia The electrocardiogram in contrast is not suggestive of infarction The *vectorcardiogram in the second row corresponds to the* ventricular extrasystoles shown in the electrocardiogram Note the closely spaced time dashes *in the early portion of the efferent* limb of the loop of the ventricular extrasystole

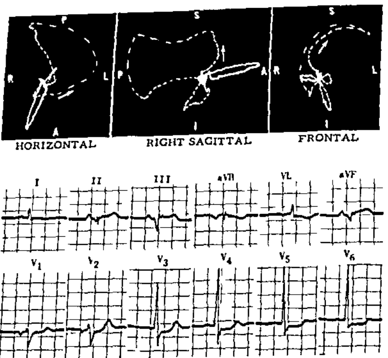


Fig 398 —Electrocardiogram and vectorcardiogram in diaphragmatic myocardial infarction of uncertain duration

In the electrocardiogram abnormal Q waves are present in lead II III and aVF

In the vectorcardiogram, recorded several days later the QRS sE loop shows marked superior displacement of the efferent limb of the loop which reverses the direction of inscription of the right sagittal planar loop and produces a crescent shaped frontal QRS loop The T sE loop is directed to the right anteriorly and slightly superiorly The vectorcardiogram is compatible with the diagnosis of diaphragmatic myocardial infarction and posterolateral ischemia

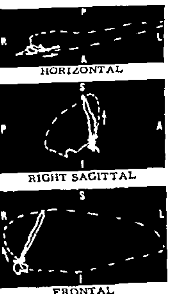


Fig 399 —Electrocardiogram and vectorcardiogram in acute diaphragmatic myocardial infarction The vectorcardiogram was recorded 4 days after the electrocardiogram and so the two records are not entirely comparable For example in the electrocardiogram the diagnostic findings consist of elevated S-T segments in leads II III and aVF and of small Q waves present in these leads In the vectorcardiogram however there is marked superior displacement of the early portion of the efferent limb of the QRS sE loop which reverses the direction of inscription of both the right sagittal and frontal planar QRS loop The T sE loop is directed almost straight superiorly Thus the electrocardiogram represents an early stage (the stage of subepicardial myocardial injury) of a developing diaphragmatic myocardial infarction while the vectorcardiogram corresponds to a somewhat later stage in the evolution of the infarction pattern in that more prominent findings of muscle necrosis and ischemia are present If an electrocardiogram had been recorded at the same time as the vectorcardiogram it would probably have shown abnormal Q waves in leads II III and aVF and isoelectric S-T segments and deeply inverted T waves in these leads

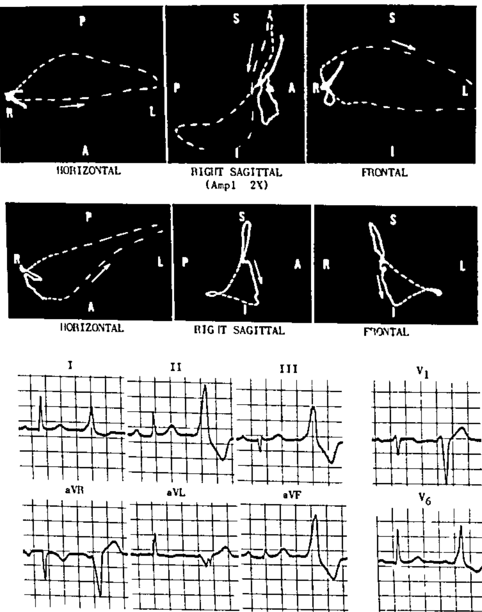


Fig 397 —Electrocardiogram and vectorcardiogram in old diaphragmatic myocardial infarction

In the electrocardiogram only leads V_1 and V_6 of the precordial leads are shown in addition to the six routine extremity leads Note that lead III displays an RSR deflection while lead aVF records an RR deflection In each of the limb leads the conducted sinus beat is followed by a coupled ventricular extrasystole

The vectorcardiogram shown in the top row corresponds to the conducted sinus beats in the electrocardiogram Note the large superiorly directed early deflection of the right sagittal QRS loop and the superior displacement of the efferent limb of the frontal QRS loop These findings plus the superior orientation of the T sÊ loop are compatible with diaphragmatic myocardial infarction and diaphragmatic ischemia The electrocardiogram in contrast is not suggestive of infarction The vectorcardiogram in the second row corresponds to the ventricular extrasystoles shown in the electrocardiogram Note the closely spaced time dashes in the early portion of the efferent limb of the loop of the ventricular extrasystole

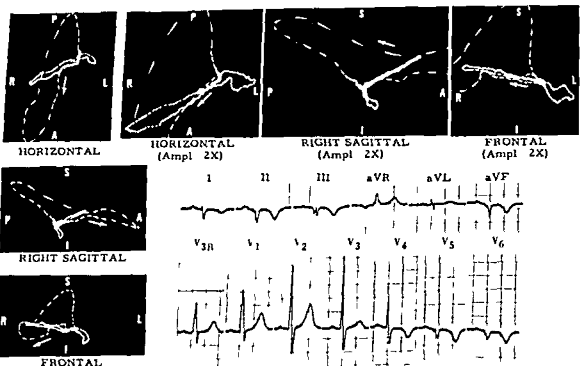


Fig 402.—Electrocardiogram and vectorcardiogram in recent posterolateral myocardial infarction. Note in the vectorcardiogram that the QRS sÊ loop is written at first anteriorly and to the right and then posteriorly, superiorly and to the right while the T sÊ loop is directed to the right and slightly anteriorly. Despite the abnormal Q waves in leads II, III and aVF of the electrocardiogram which would suggest diaphragmatic myocardial infarction, the vectorcardiogram shows no evidence of the lesion but is diagnostic of posterolateral infarction and posterolateral myocardial ischemia.

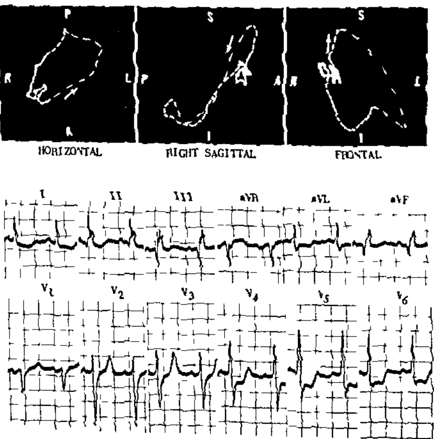
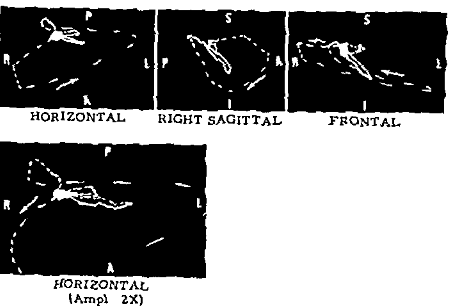


Fig 400 —Electrocardiogram and vectorcardiogram in old diaphragmatic myocardial infarction Note the marked S T segment depression in leads V through V of the *electrocardiogram* and the corresponding S-T vector in the vectorcardiogram which is directed primarily to the right The S-T segment deviation and the S-T vector in the vectorcardiogram are compatible with anterolateral subendocardial myocardial injury



I II III aVR aVL aVF

V_1 V_2 V_3 V_4 V_5 V_6

Fig 401 —Electrocardiogram and vectorcardiogram in old healed posterolateral myocardial infarction Note the abnormal Q waves in leads V and V_6 and the wide tall initial R wave in lead V_1 of the electrocardiogram Corresponding findings in the vectorcardiogram consist of the large rightward and anterior early deflection of the QRS sE loop

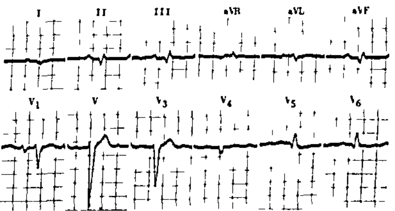
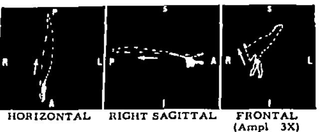


Fig 405 —Electrocardiogram and vectorcardiogram in old localized diaphragmatic anterolateral (or apical) myocardial infarction



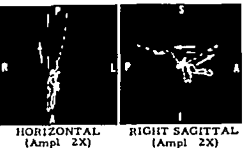


Fig 406 —Electrocardiogram and vectorcardiogram in old diaphragmatic anterolateral (apical) myocardial infarction Note that abnormal Q waves are recorded by lead I II III aVF and V through V of the electrocardiogram In the vectorcardiogram the efferent limb of the QRS sE loop is displaced markedly posteriorly superiorly and medially Thus the directions of inscription of the horizontal and frontal QRS loops are reversed Consequently both the electrocardiogram and the vectorcardiogram are diagnostic of combined diaphragmatic anterolateral or apical myocardial infarction

Fig 403 —*Electrocardiogram* and *vectorcardiogram* in old healed diaphragmatic anterolateral myocardial infarction Note the initial small R waves in leads II III and aVF which are related to the initial inferior inscription of the sagittal and frontal QRS loops Thus the diagnosis of diaphragmatic infarction might be missed in the electrocardiogram while the *vectorcardiogram is clearly diagnostic* of diaphragmatic anterolateral infarction

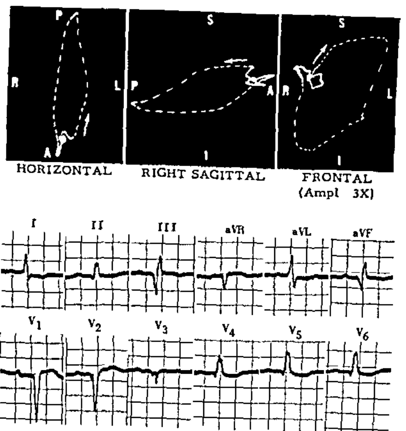


Fig 404 —Electrocardiogram and vectorcardiogram in old healed diaphragmatic anteroseptal myocardial infarction

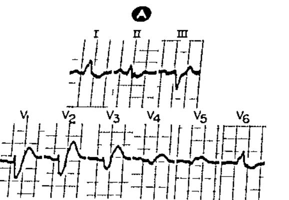


Fig 410 —A electrocardiogram strongly suggestive of acute septal infarction complicating left bundle branch block The initial R waves diminish in amplitude from right to left across the precordium and the S-T segments are elevated in leads V and V B electrocardiogram strongly suggestive of acute diaphragmatic myocardial infarction complicating left bundle branch block In this instance the diagnosis of acute infarction rests primarily on the elevated S-T segments in leads II III and aVF

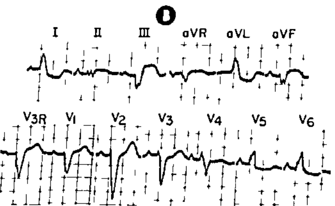


Fig 411 —Electrocardiogram and vectorcardiogram in left bundle branch block with old septal infarction Note in the electrocardiogram the small Q waves in leads I aVL, V and V The corresponding finding in the vectorcardiogram is the rightward inscription of the initial portion of the QRS sE l op

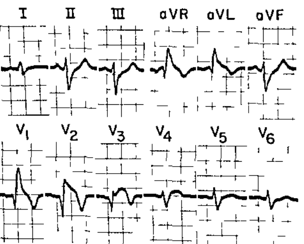


Fig 407 —Acute anteroseptal myocardial infarction with right bundle branch block Note that the distinctive electrocardiographic features of both anteroseptal infarction and right bundle branch block are clearly evident

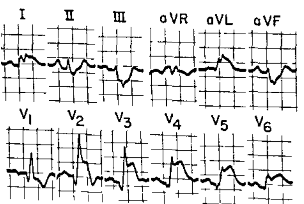


Fig 408 —Acute extensive anterior myocardial infarction with right bundle branch block

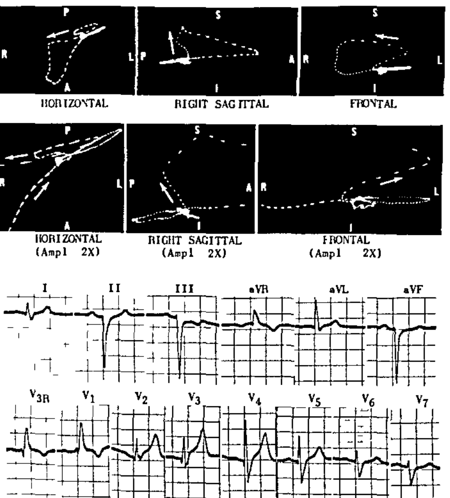
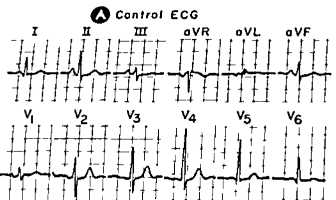
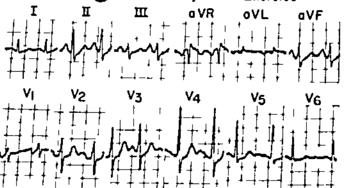


Fig 409 —Electrocardiogram and vectorcardiogram in old anterior myocardial infarction with right bundle branch block

In the electrocardiogram the main finding suggestive of old anterior infarction is the presence of Q waves in leads V_4 through V_6 although this is rather equivocal evidence for the diagnosis of infarction In the vectorcardiogram however there is posterior displacement of the efferent limb of the QRS sÊ loop while the remaining portion of the QRS *sÊ loop is diagnostic* of the variant type of right bundle branch block The posterior shift in the efferent limb of the horizontal QRS loop is diagnostic of old anterior myocardial infarction





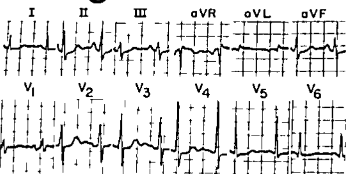


Fig. 414.—An equivocal electrocardiographic response to the double Master stress test. The control electrocardiogram (**A**) is within normal limits. The record made immediately after exercise (**B**) shows a sinus tachycardia and apparent depression of the S–T segments in all leads except aVR, aVL and V_1. The S–T segment depression persists, although less marked, at 2 minutes after exercise, as noted in **C**. Ten minutes after exercise the electrocardiogram had returned to normal. There is a strong suspicion that the S–T segment depression in **B** may be related to the effects of tachycardia on the depth of the atrial T wave. Thus, if one were to continue the T–R segment through the QRS deflection, it would be found to intersect with the S–T segment at the junction (J) in most leads. This would suggest that the S–T segment depression is actually produced by a superimposed deep atrial T wave. The above three electrocardiograms illustrate some of the difficulties which may be encountered in attempting to evaluate the significance of the electrocardiographic response to exercise.

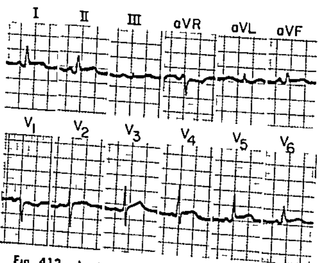
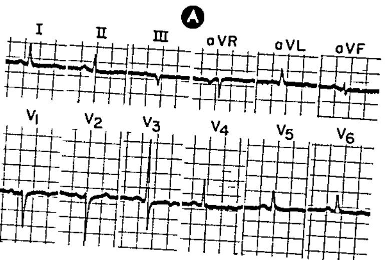


Fig 412 –Acute pericarditis Note the elevated S–T segments in leads I II aVF and V through V



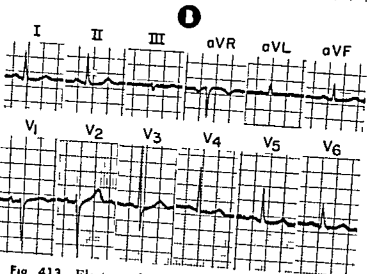


Fig 413 –Electrocardiographic findings in a woman 49 with myxedema treated with thyroid **A** record from the patient before administration of thyroid Note the low upright or flat T waves in the limb leads and leads V_5 and V the inverted T wave in lead V_1 and the diphasic T wave in lead V Shortly after record **A** was obtained the patient was started on 15 mg thyroid daily **B** electrocardiogram recorded 1 month later There is a general increase in T wave amplitude and the T waves which previously were diphasic or inverted have become upright

C 2 Minutes After Excercise

I II III aVR aVL aVF

V_1 V_2 V_3 V_4 V_5 V_6

D 4 Minutes After Excercise

I II III aVR aVL aVF

V_1 V_2 V_3 V_4 V_5 V_6

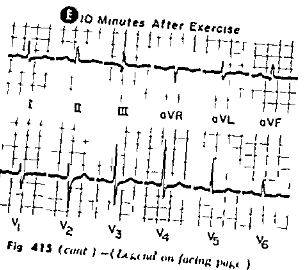


Fig 413 *(cont)* —(*Legend on facing page*)

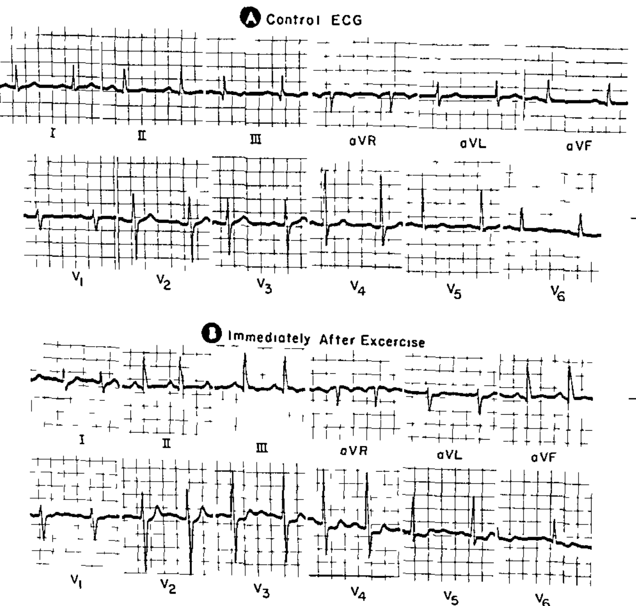


Fig 415 —A positive electrocardiographic response to the double Master stress test. The control electrocardiogram **(A)** is within normal limits while the electrocardiograms taken immediately after exercise *(B)* and 2 minutes after exercise **(C)** show widespread T wave changes and S–T segment deviation. In record **D** made 4 minutes after exercise inverted T waves persist in leads I and V through V_4. Record **E** obtained 10 minutes after exercise has virtually returned to normal.

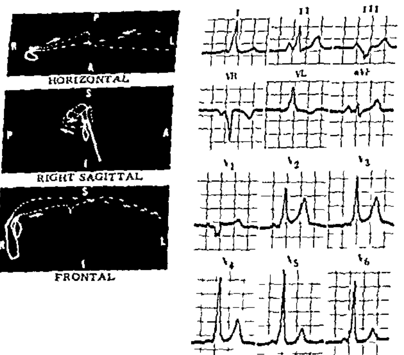


Fig 418 — Electrocardiogram and vectorcardiogram in the Group B ventricular pre-excitation pattern

HORIZONTAL (Ampl 2X) RIGHT SAGITTAL (Ampl 2X) FRONTAL (Ampl 2X)

HORIZONTAL

RIGHT SAGITTAL

FRONTAL

I II III VR aVL VF

V1 V2 V3 V4 V5 V6

Fig 419 — Electrocardiogram and vectorcardiogram in the Group B ventricular pre-excitation pattern

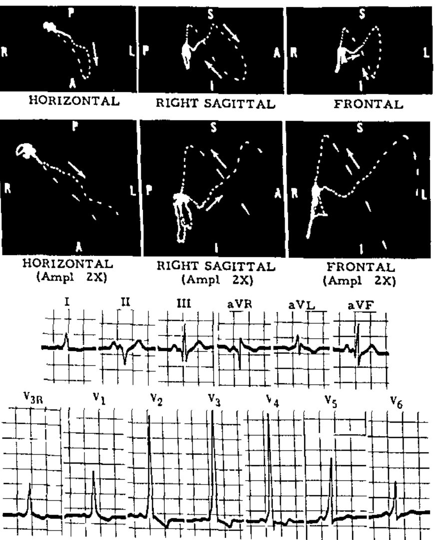


Fig 416 —Electrocardiogram and vectorcardiogram in the Group A ventricular pre excitation pattern

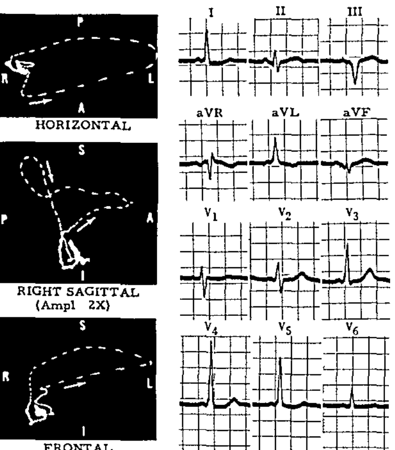


Fig 417 —Electrocardiogram and vectorcardiogram in the Group B ventricular pre excitation pattern

Fig 422 —Sinus arrhythmia of minor degree in two different patients



Fig 423 —Upper atrioventricular nodal rhythm with prolonged antegrade conduction or alternatively coronary sinus rhythm Retrograde P waves precede each ventricular deflection by a P–R interval of about 0.17–0.18 second and are inverted in leads II III and aVF and upright in lead aVR Typically in so called upper atrioventricular nodal rhythm without antegrade first degree atrioventricular block the P–R interval is 0.12 second or less and so prolonged forward or antegrade conduction of the atrioventricular nodal impulse would have to be assumed in this record or as an alternative explanation it might be postulated that the site of impulse origin is in the coronary sinus region near the atrial end of the atrioventricular node Impulses arising in this area would be propagated in a retrograde direction through the atria just like atrioventricular nodal impulses but unlike the latter would have to traverse the entire length of the atrioventricular junction and thus just as if they originated in the sinoatrial node — hence the longer P–R interval The two possibilities cannot usually be distinguished

Fig 420 –Electrocardiogram and vectorcardiogram in the Group B ventricular *pre excitation pattern*

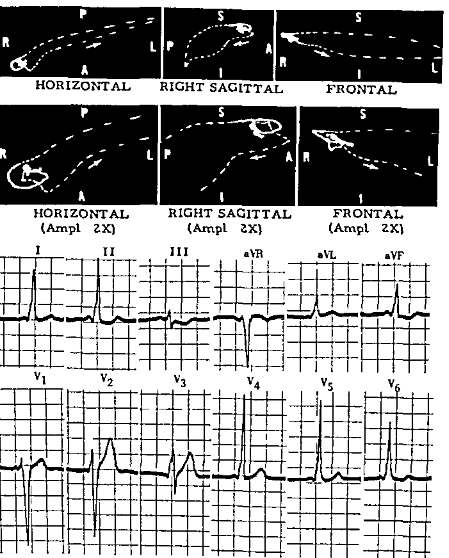


Fig 421 –Electrocardiogram and vectorcardiogram in the Group B ventricular pre excitation pattern

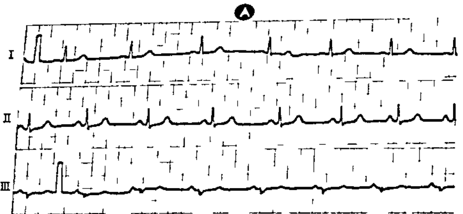


Fig 422 —Sinus arrhythmia of minor degree in two different patients



Fig 423 —Upper atrioventricular nodal rhythm with prolonged antegrade conduction or alternatively coronary sinus rhythm Retrograde P waves precede each ventricular deflection by a P–R interval of about 0 17–0 18 second and are inverted in leads II III and aVF and upright in lead aVR Typically in so-called upper atrioventricular nodal rhythm without antegrade first-degree atrioventricular block the P–R interval is 0 12 s cond or less and so prolonged forward or antegrade conduction of the atrioventricular nodal impulse would have to be assumed in this record or as an alternative explanation it might be postulated that the site of impulse origin is in the coronary sinus region near the atrial end of the atrioventricular node Impulses arising in this area would be propagated in a retrograde direction through the atria just like atrioventricular nodal impulses but unlike the latter would have to traverse the entire length of the atrioventricular junctional tissues just as if they originated in the sinoatrial node—hence the longer P–R interval These two possibilities cannot usually be distinguished

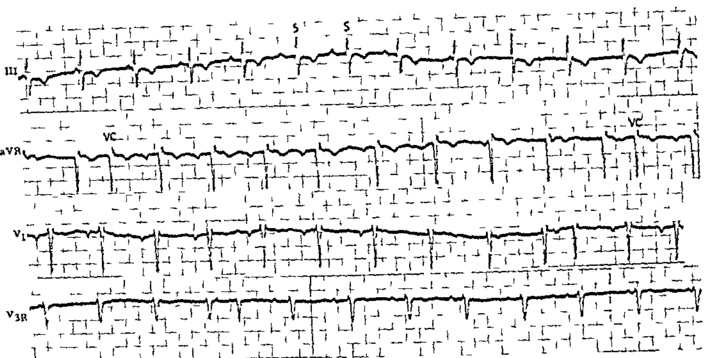


Fig 424 —Intermittent atrioventricular nodal rhythm with incomplete atrioventricular dissociation and frequent ventricular capture beats (VC) recorded in a youth 16 with acute rheumatic fever Note the relatively rapid rate of the atrioventricular nodal rhythm Conducted sinus beats are labeled S in lead III

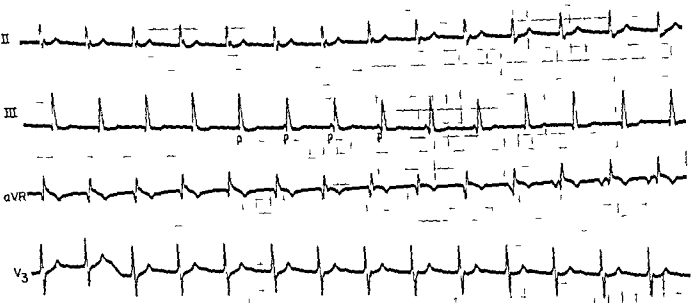
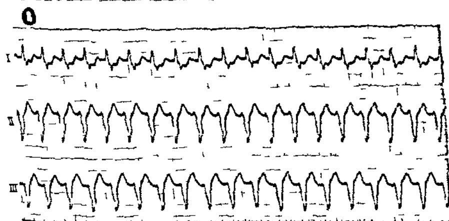
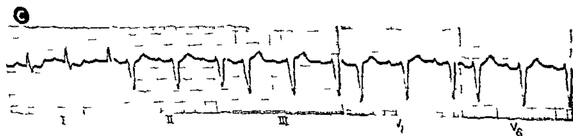


Fig 425 —Atrioventricular nodal rhythm with complete atrioventricular dissociation due to equalization of the atrial and ventricular rates In lead III some of the sinus P waves have been indicated by the symbol P

Fig 426.—A [illegible] rhythm [illegible] (with [illegible] of the QRS [illegible]) [illegible] Sinus [illegible] electrocardiogram A the QRS interval of the conducted sinus beats (*SB*) in the [illegible] strips is approximately 0.13 second in leads II, III and aVI. This intraventricular conduction disturbance makes it difficult to localize the site of origin of the escape rhythm appearing intermittently in these strips. There is intermittent complete atrioventricular dissociation; the QRS duration of the escape beats (*E*) is approximately 0.16 second and their appearance differs strikingly from the QRS configuration of the conducted sinus beats. At the onset and offset of the escape rhythm, ventricular fusion beats (*FB*) can be seen, but it is debatable whether or not this finding necessarily means that the escape rhythm arises in the ventricles. The rate of the escape rhythm is 80 beats per minute, which is far faster than the rates observed in the usual ventricular escape rhythm. In record **B**, from the same patient, coupled premature beats (*PC* and *PCs*), probably of atrioventricular nodal origin, can be seen, and these are frequently followed by a short period of escape rhythm. The ventricular complexes during these periods of escape rhythm are greatly widened and of aberrant configuration in some instances, but in lead III escape beats can be identified which are identical in appearance to the conducted sinus beats, suggesting that the escape rhythms in **B** and in **A** both originate in the atrioventricular node. Despite this fact, ventricular fusion beats can be identified in **B** just as in **A**.







ient had returned to normal sinus rhythm
duration as those present during the parox
I B is obviously of supraventricular (prob
f the QRS deflections during the tachycar

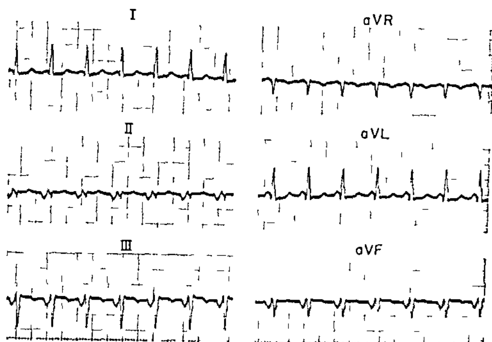


Fig 439 —Paroxysmal atrioventricular nodal tachycardia Note the inverted retrograde P waves preceding the ventricular deflections in leads II III and aVF and the upright retrograde P waves in lead aVR

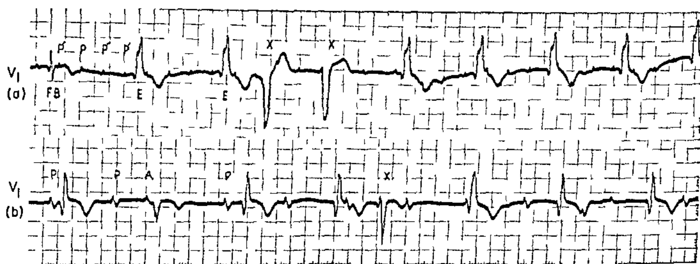


Fig 440 —Both strips of lead V_1 were recorded from the same patient the second record having been made about 24 ho ...) with ... as are ... ll represent conducted atrial beats while the first ventricular deflection in the lead strip is probably a ventricular fusion beat (*FB*) In lead V_1 (*b*) the atrial rhythm is of sinus node origin (sinus P waves are labeled *P*) There is almost complete atrioventricular block and the ventricular rhythm arises in an idioventricular pacemaker The P wave designated A is an atrial premature beat and the QRS deflection following it is probably a ventricular fusion beat The ventricular complex labeled X appears to be a conducted sinus beat

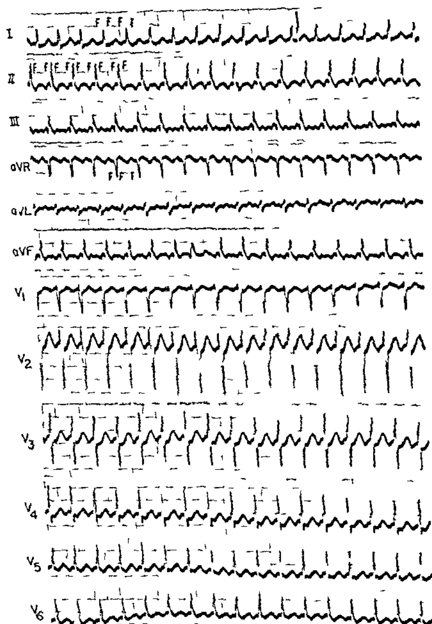


Fig 441 —Atrial flutter with 2 1 atrioventricular response The atrial flutter waves are labeled F

I

II

III

F F F F F F F F F

V_1

V_3

Fig 442 —Atrial flutter approaching fibrillation (Key *F* atrial flutter waves)



Fig 443 —Atrial flutter with widely varying atrioventricular response and ventricular aberration of some of the QRS deflections (Key *F* atrial flutter waves)

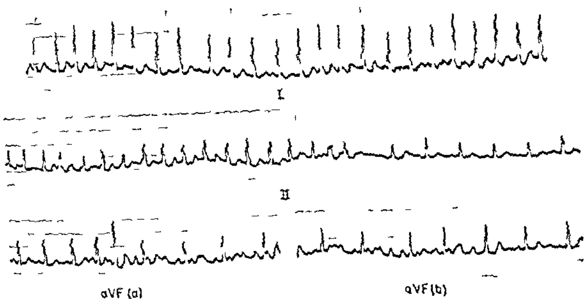


Fig 444 —Impure atrial flutter with spontaneous transition to and from sinus rhythm at end of lead II first part of lead aVF (a) and latter part of lead aVF (b)



Fig 445 —Atrial fibrillation.

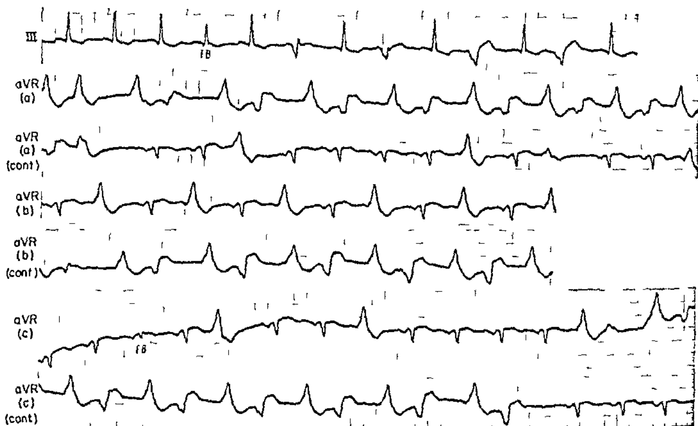


Fig 446 —Alternating bidirectional ventricular tachycardia Shown are three continuous strips of lead aVR and a single strip of lead *III* containing periods of alternating bidirectional tachycardia The deflections labeled *FB* are ventricular fusion beats The presence of ventricular fusion beats the marked widening and distortion of the QRS deflections and the presence of atrioventricular dissociation are all compatible with a ventricular origin of the ectopic beats



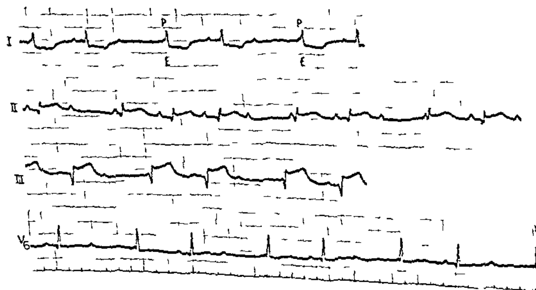
Fig
ventrici
proachu
less and less clear cut In lead II (c) the ventricular tachycardia, shortly before its termination consists of intermittent short paroxysms of tachycardia between which are interspersed one to five ventricular beats of an atrioventricular nodal escape rhythm In lead II (d) there is a trigeminal rhythm, each group of three ventricular beats being composed of an atrioventricular nodal beat followed by two ventricular extrasystoles There is also atrioventricular dissociation In lead II (e) there is an atrio entricular nodal rhythm with complete atrioventricular dissociation

Fig 448 (*left*) —Lead strips recorded from the same patient during a 2 hour period prior to his death In lead II (*a*) there is a sinus tachycardia with a 1 1 atrioventricular conduction and slightly prolonged intraventricular conduction In the next strip of lead V atrial deflections cannot be identified and so the rhythm is either atrioventricular nodal or idioventricular in origin In lead II (*b*) there is marked prolongation of the QRS interval and the QRS complexes are bizarre in appearance The ventricular beats presumably arise in an idioventricular pacemaker In lead II (*c*) there is a relatively slow ventricular tachycardia while in lead II (*d*) the rhythm is ventricular flutter Finally in lead II (*e*) ventricular fibrillation appears

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Fig 449 (*below*) —Complete atrioventricular block with progressive deterioration of pacemaking [illegible] ity in the idioventricular center the ventricular rate gradually slows and the QRS deflections progressively widen

escape beat and the second a conducted [illegible]
sinus beats which are conducted into the ventricles are labeled *P*. The remaining P waves [illegible]
impulses which are not conducted into the ventricles



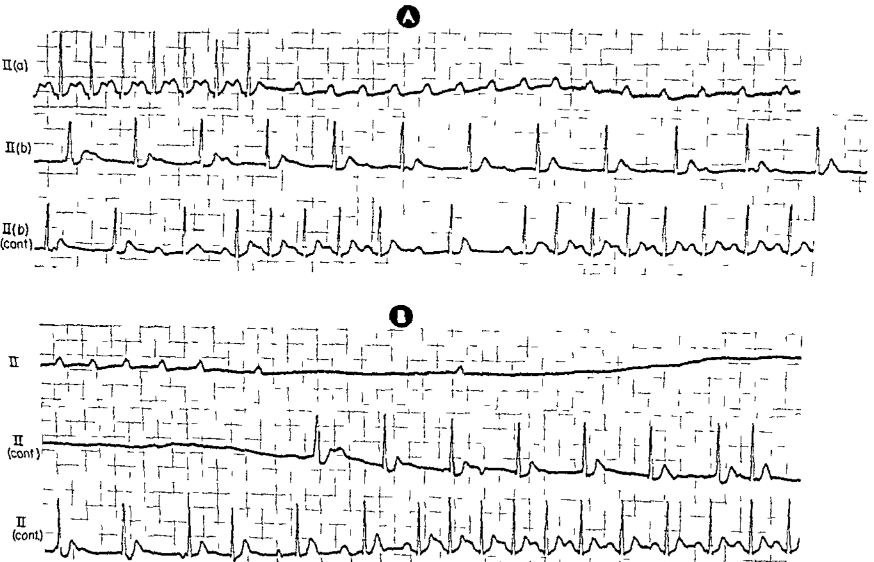


Fig 452 —Lead strips recorded from the same patient on several occasions during Stokes Adams attacks In **A** there can be seen rapid sinus tachycardia with onset of complete atrioventricular block and prolonged ventricular standstill and finally appearance of atrioventricular nodal escape rhythm In **B** much the same sequence of events can be seen except that here there is a long period of atrial as well as ventricular standstill (*Continued*)

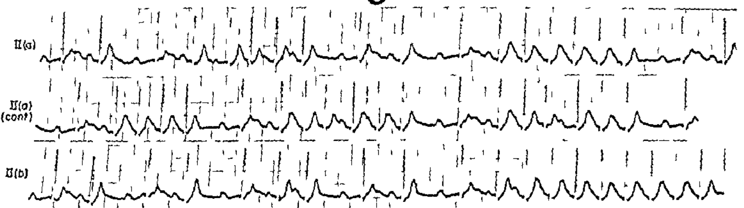




Fig 452 (*cont*) —In **C** there is rapid sinus tachycardia with short period of 2:1 atrioventricular block. In **D** the first strip shows complete atrioventricular block while the second strip shows almost complete atrioventricular block and dissociation with occasional ventricular captures.

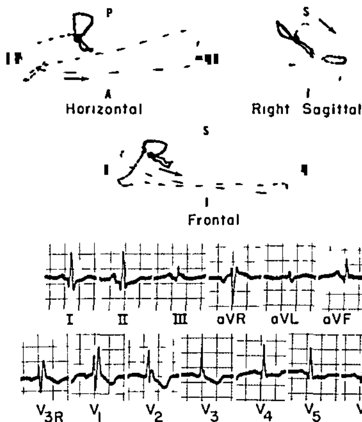


Fig 453 —Electrocardiogram and vectorcardiogram *in right bundle branch block* and old healed posterolateral myocardial infarction

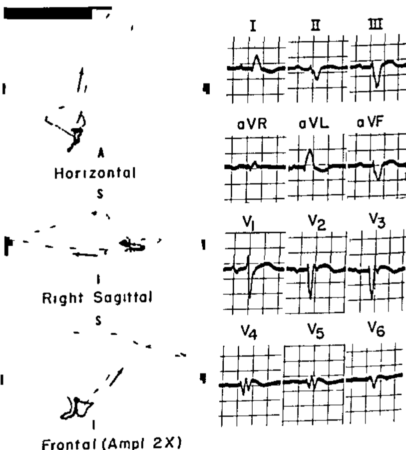
In the electrocardiogram note the relatively deep Q wave in lead I and the relatively tall initial R wave in lead V_1 These findings are somewhat suggestive of an old posterolateral infarction but they cannot be construed as being diagnostic of this condition

In the vectorcardiogram there is a large early deflection of the QRS sÊ loop to the right and anteriorly and the efferent limb *of the loop is displaced anteriorly These* findings are strongly suggestive of old pos*terolateral myocardial infarction*

The features of right bundle branch block are equally obvious in both the electrocardiogram and vectorcardiogram

Fig 454 —Electrocardiogram and vectorcardiogram in left bundle branch block with recent infarction of the anterolateral wall of the left ventricle and interventricular septum Autopsy *confirmed the recent infarction of the lateral* wall of the left ventricle and interventricular septum but also demonstrated recent infarction of the posterior wall of the left ventricle multiple healed infarcts of the entire left ventricle and dilatation and hypertrophy of the heart predominantly of the left ventricle

In the electrocardiogram the QRS duration is 0 14 second and there are broad slurred R waves in leads I and aVL Additional findings which indicate septal and anterolateral infarctions superimposed on left bundle branch block are small Q waves in leads I and aVL relatively tall R wave in lead V with diminishing R wave amplitude from right to left W shaped ventricular deflections in leads V and V and QS deflection in lead V_6 There is minimal elevation of the S–T segments in leads V through V



tive of coexisting septal and anterolateral infarction consist of the following large early deflection of the QRS sÊ loop anteriorly and only

the right and inferiorly and the T sÊ loop is 180 discordant to the QRS sÊ loop

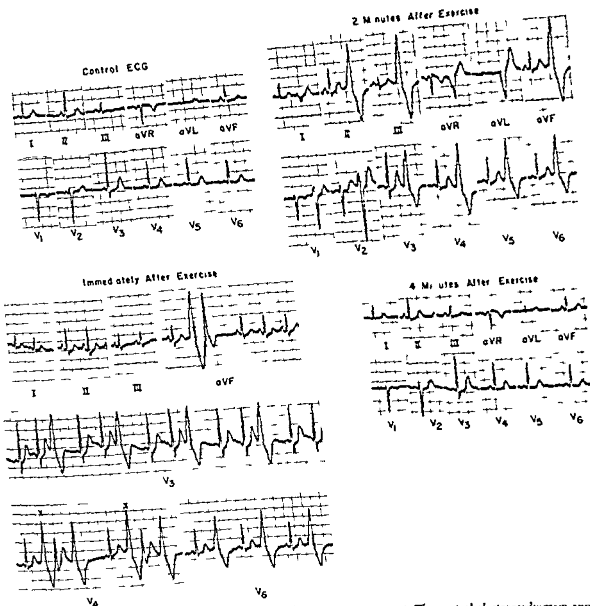


Fig 455 –Positive electrocardiographic response to the Master exercise test The control electrocardiogram appears to be entirely within normal limits In the record made immediately after exercise moderate depression of the S–T segments c … and V through V A more striking finding in this electrocardiogram is the appearance of … constitutes eq … cal evidence for interpreting … lar extrasystole in the postexercise tracings is in itself diagnostic of a positive response

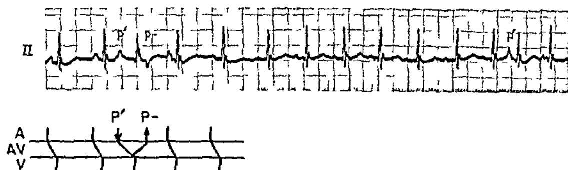


Fig 456 —*Reciprocal atrial beat following* an atrial extrasystole (Key *P* premature atrial extrasystoles *P—* reciprocal P wave following the first ectopic atrial beat) The explanation for reciprocal ventricular beats has been discussed in the text and in Figure 246 The same mechanism operating in a different direction can explain the presence of a reciprocal atrial beat Thus the first ectopic atrial beat is transmitted through the atrioventricular node slowly some *fibers of the atrioventricular conducting pathways being entirely refractory to the ectopic beat* Shortly before emerging from the atrioventricular node the ectopic atrial impulse splits one impulse continuing into the ventricles and the other being propagated back up the atrioventricular node into the atria by the previously refractory conducting pathways

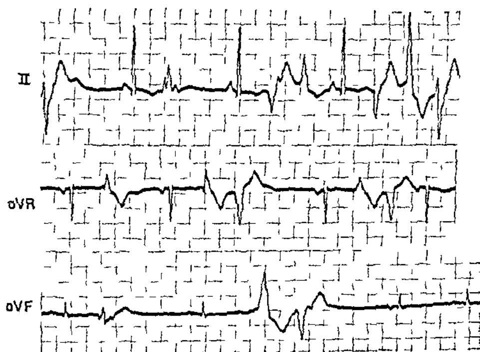


Fig 457 —Multifocal and frequently bidirectional ventricular extrasystoles occurring in a patient intoxicated with digitalis

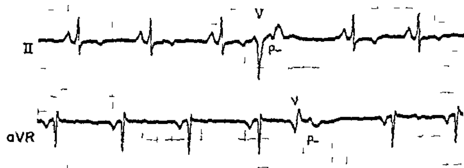


Fig 458 —
the retrograde
and interrupt t
ular extrasystoles

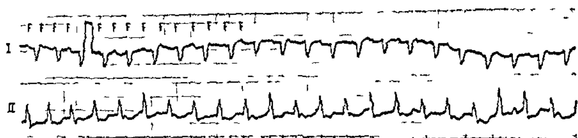


Fig 459 —Simultaneous atrial flutter and atrioventricular nodal tachycardia with probably complete atrioventricular dissociation In both lead strips atrial flutter (F) waves can be identified they appear at a rate of 250 per minute with a rhythm which is independent of the ventricular rhythm The ventricular complexes appear at a rate of 160 per minute *and exhibit essentially regular cycles Thus* there must be complete atrioventricular dissociation between the atria and the ectopic atrioventricular nodal pacemaker This combination of rapid rhythms occurred in a patient with digitalis intoxication and is a rarely observed type of arrhythmia.

I

III

P P P P P P P P P P

aVL

aVF

Fig 460 —*Intermittent paroxysmal* ventricular tachycardia with atrioventricular dissociation occurring in the presence of first-degree atrioventricular block. Sinus P waves (*P*) can be identified *superimposed* on the S–T segment or T waves *of the ectopic* ventricular beats of the paroxysmal tachycardia

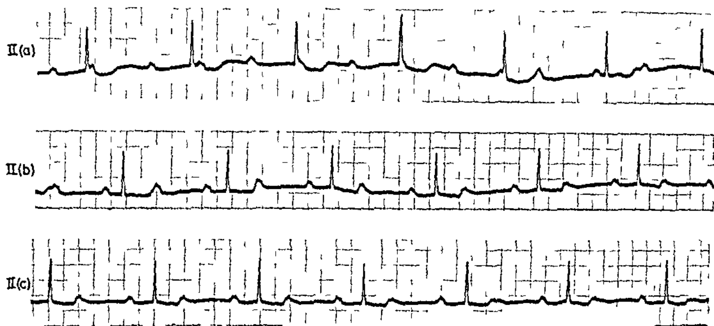


Fig 461 — The lead strips shown ar ... ought that ... However ... plete and that the R–R intervals in ... the ventricular rhythm must have ... nodal pacemaker

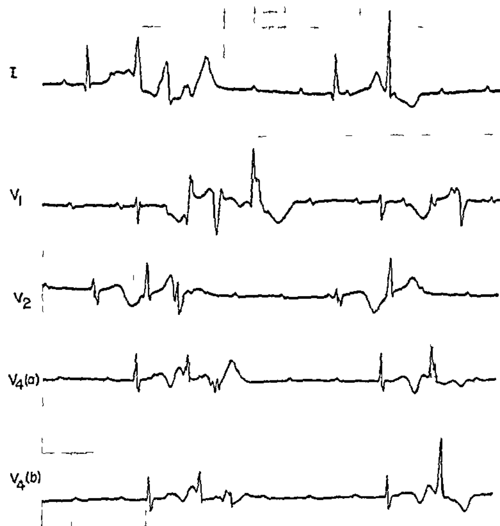


Fig 462 — Multifocal ventricular extrasystoles occurring in the presence of complete atrioventricular block Note that the ventricular extrasystoles are bidirectional in some of the leads

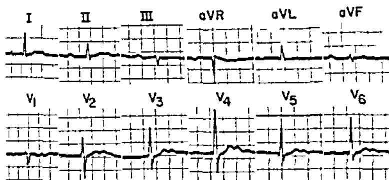
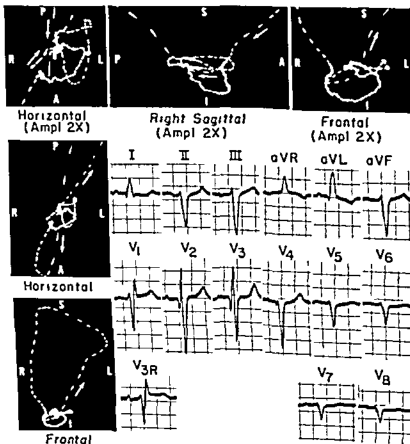


Fig 463 — Hypokalemia This electrocardiogram is compatible with the diagnosis of hypokalemia in that it displays strikingly prominent U waves in virtually all leads slightly depressed S–T segments in leads V through V and low T waves in most leads An additional and probably unrelated finding is the presence of first-degree atrioventricular block



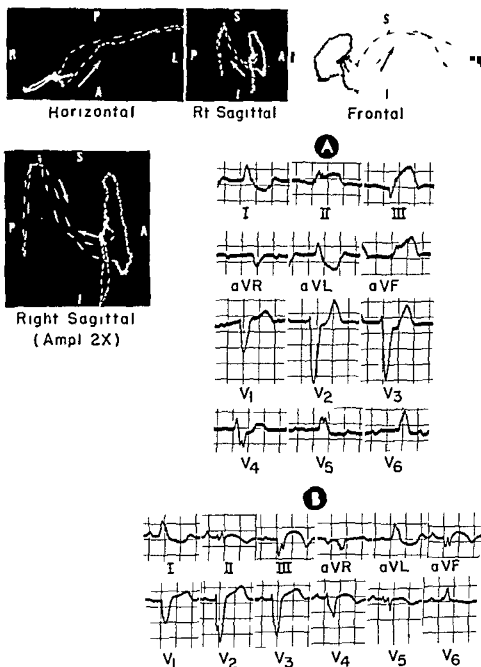


Fig 465 —Electrocardiogram and vectorcardiogram in left bundle branch block with acute diaphragmatic myocar

loops point of origin while the T sE loop is large and is dir
graphic features of left bundle branch block are difficult to
normal direction of the initial portion of the QRS sE loop to the left and anteriorly The above vectorcardiogram is therefore compatible with left bundle branch block and diagnostic of diaphragmatic myocardial ischemia subepicardial injury and infarction

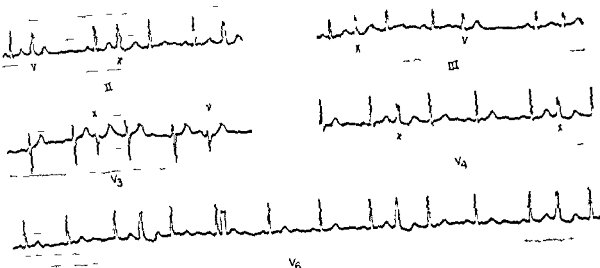


Fig 466 —Interpolated ventricular extrasystoles The ectopic ventricular beats labeled X are interpolated while those labeled V are not

I
240 = 120 X 2
118
238 119 X 2
236 = 118 X 2
II
366 = 122 X 3
238 = 119 X 2
240 = 120 X 2
240 = 120 X 2
119
240 = 120 X 2
363 = 121 X 3
V6

Fig 467 —Atrioventricular nodal parasystole The ectopic ventricular beats are less *than 0 12* second in duration they show varying coupling intervals and yet their interectopic intervals are a whole *number multiple* of approximately 1 20 seconds

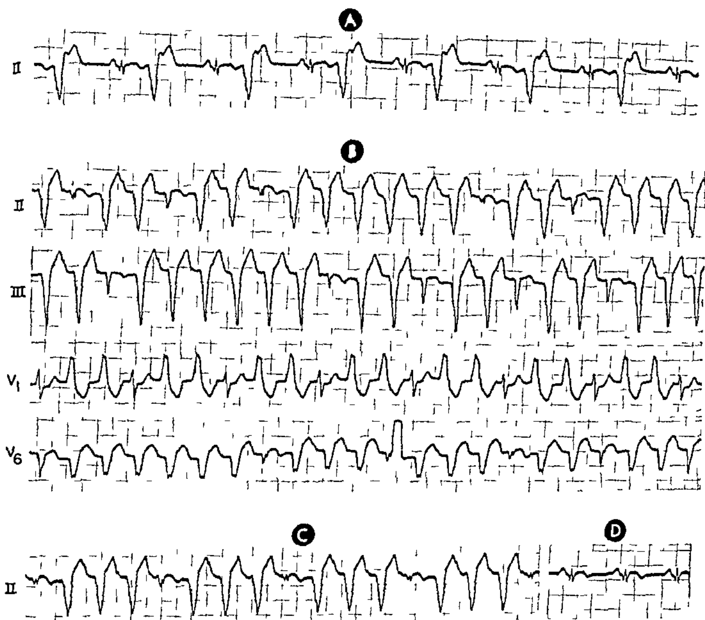


Fig 468 —Ventricular tachycardia In **A** the strip of lead II shows a coupled rhythm with each conducted sinus beat being followed by a ventricular extrasystole In **B** the lead strips of both leads II and III show ventricular extra

of the ectopic ventricular beats

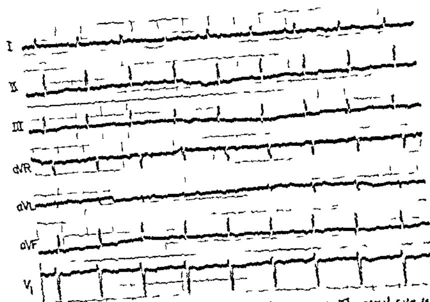


Fig 469 —Paroxysmal atrial tachycardia with 2 1 atrioventricular response The atrial rate is approximately 175 beats per minute

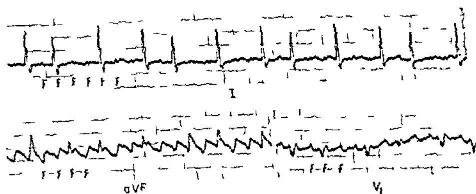


Fig 470 Atrial flutter (F) with predominantly 2 1 and 3 1 atrioventricular response

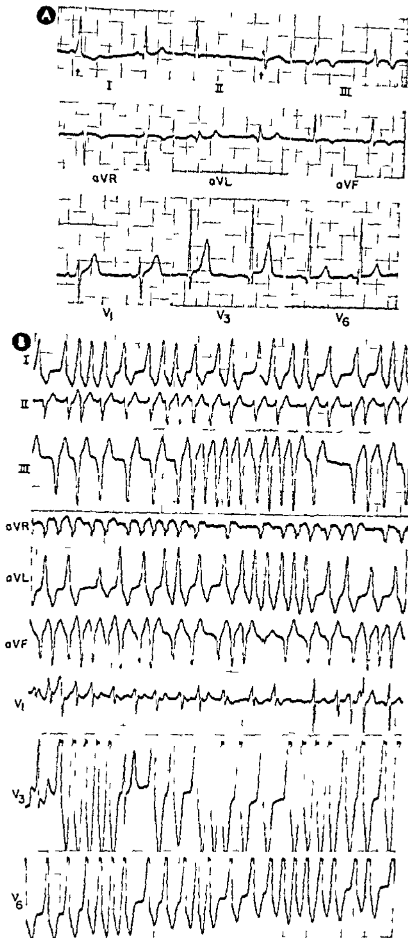


Fig 471 —Wolff Parkinson White syndrome and rapid atrial fibrillation mimicking ventricular tachycardia Electrocardiogram **A** shows a sinus rhythm with predominantly normal intraventricular conduction However the first ventricular complex in lead I shows a short P–R interval a slurred upstroke of the R wave (delta wave indicated by an arrow) and an inverted T wave while the second QRS deflection in lead II shows a minute embryonic R wave followed by the main upward component of the QRS deflection These two findings particularly the former are strongly suggestive of intermittent ventricular pre excitation Electrocardiogram **B** was recorded from the same patient about 8 months later At first glance the rapid ventricular rate the widened bizarre QRS deflections and the marked secondary ST–T changes would all suggest ventricular tachycardia if one were not aware of the findings in record **A** However the marked irregularity of the ventricular rhythm in **B** the marked slurring of the early part of the upstroke of the R waves in leads I aVL V_1 V and V and the corresponding slurring of the downstroke of the QS deflections in leads III aVR and aVF suggest the possibility that the rhythm represents atrial fibrillation with rapid ventricular response and ventricular aberration due to pre excitation Electrocardiogram **C** recorded from the same patient after conversion of the atrial fibrillation to sinus rhythm shows a typical example of Group A ventricular pre excitation

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It is probable that at least half of the oral dose remains in the lumen of or in the organs excreting into the intestinal tract. Only 10 per cent appears in the urine. About half of the amount injected subcutaneously passes into the urine. The remainder must be metabolized. Effective therapeutic blood levels are 0.5 to 1.0 mg. per cent. The ion is excreted by the kidney mainly through glomerular filtration.

Hydrazines.—Figures 149 and 150 show the excretion rates of 1-hydrazinophthalazine. Only about 1 or 2 per cent appears in the urine whether ingested or injected. The method of measuring the drug depends upon the presence of the hydrazine moiety; what happens to the remaining 98 per cent is unknown.[3] About half of the urinary hydrazine is loosely bound to sulfhydryl groups, mainly cysteine. Obviously, doses could be given at longer intervals were it not for the possibility of reactions following single large doses.

LIMITATIONS OF THE METHOD

There are no known limitations of the method in so far as hypertension itself is concerned. The ages of the patients treated have ranged from seven to seventy-nine years. Blood pressure *per se* can be lowered significantly in all patients in whom it is elevated due to generalized vasospasm. In the long term, however, there are certain limits of effectiveness against the hypertensive process which we have observed in over 250 cases. In general the method does not prolong life when renal disease is advanced, when cerebral arteriosclerosis is severe, and when pathological changes in the vessels themselves have so progressed that the occurrence of lesions incompatible with life is imminent. Hyphex is effective, however, in cases so advanced as to be unsuitable for surgery on the sympathetic nervous system, in children with severe renal diseases and malignant hypertension, in eclampsia and toxemias of pregnancy, and even in cases of congestive heart failure in subjects appearing in almost terminal states.

Uremia and Renal Insufficiency.—Organic renal disease, primary or secondary, of such an advanced nature as to cause uremia responds little or not at all to Hyphex. The blood pressure may be moderately lowered without adverse results, the element of renal vasospasm partly combatted, but the progress of the disease is at the most slowed, postponing the inevitable outcome for a few weeks or months. Uremia therefore constitutes one limit of effectiveness for Hyphex, as might be expected, unless the progress of the renal damage is relatively static.

Moderate nitrogen retention (NPN up to 50 or 60 mg. per cent)* may remain when the blood pressure is carefully lowered and after several months may regress, an unexpected phenomenon. Slight nitrogen retention (NPN 26 to 35 mg. per cent) may disappear completely with the establishment of normotension for several months. When the NPN has been consistently much over 60 mg. per cent, a fall is not to be expected (Table 91).

Cerebral Arteriosclerosis and Thrombosis.—It is extremely doubtful that Hyphex prevents the occurrence of thromboses in cerebral arteries severely

* By the method used in this hospital, 25 mg. per cent is the upper limit of normal.

d m ned by arteriosclerosis (Table 92) Two and possibly three episodes
ed during induction of control of

sive levels three of these had suffered p

1 A fifty-eight year-old woman had exhibited neurogenic hypertension for fifteen years She suffered from an attack of hemiplegia six months he recovered almost completely
erse symptoms An attack of
nsion for one week four months after she had been taking Hypnex al hemiplegia recurred, recovery lowly took place in three weeks It is probable that a small thrombosis developed during the hypotensive period

T BLE 91 —CHAN ES IN N N PROTEIN NIT EN I BLO I DUCED B HYPH X

(MALI NT HYPERTENS N)

Cha g NPN mg per ent	I itial 5-30*	N protei it ogen mg per ent 31 40	1 50	51 60	61 80	O er 80	S b-total	Total
N Case	10	11	9	1	5	4		40
Red ced to Normal (less than 25)	4	3	2				9	
Red ed by 10-20			2	1		1	6	
Red ced by 21 30					1	1	2	
Red ced by 130						1	1	18 Reduced
I ased by 10-20	1		1		1		3	
I eased by 21 30	1						1	
I c eased by 41 50			1				1	5 Incr ased
U hanged ±10	4	8	3		1	1	1	17 U hanged
								40

PSP l ss than 1 per cent 15 n.

appeared Six months later he suffered a slow thrombosis of an artery in his pons eight months later a second similar cerebral accident and ten months later a third from which he died The onset of each was gradual and there was never blood in his spinal fluid Cerebral arterial thrombosis was the probable cau e of each episode

3 A thirty three-year-old woman had undergone lumbodorsal sympathectomy four years previously for malignant hypertension The malignant stage recurred and she was given Hyplex Ten months later she suffered a thrombosis of a cerebral vessel which left her partly paralyzed

It is probable that at least half of the oral dose remains in the lumen of or in the organs excreting into the intestinal tract. Only 10 per cent appears in the urine. About half of the amount injected subcutaneously passes into the urine. The remainder must be metabolized. Effective therapeutic blood levels are 0.5 to 1.0 mg. per cent. The ion is excreted by the kidney mainly through glomerular filtration.

Hydrazines.—Figures 149 and 150 show the excretion rates of 1-hydrazinophthalazine. Only about 1 or 2 per cent appears in the urine whether ingested or injected. The method of measuring the drug depends upon the presence of the hydrazine moiety; what happens to the remaining 98 per cent is unknown.[3] About half of the urinary hydrazine is loosely bound to sulfhydryl groups, mainly cysteine. Obviously doses could be given at longer intervals were it not for the possibility of reactions following single large doses.

LIMITATIONS OF THE METHOD

There are no known limitations of the method in so far as hypertension itself is concerned. The ages of the patients treated have ranged from seven to seventy-nine years. Blood pressure *per se* can be lowered significantly in all patients in whom it is elevated due to generalized vasospasm. In the long term, however, there are certain limits of effectiveness against the hypertensive process which we have observed in over 250 cases. In general the method does not prolong life when renal disease is advanced, when cerebral arteriosclerosis is severe, and when pathological changes in the vessels themselves have so progressed that the occurrence of lesions incompatible with life is imminent. Hyphex is effective, however, in cases so advanced as to be unsuitable for surgery on the sympathetic nervous system, in children with severe renal diseases and malignant hypertension, in eclampsia and toxemias of pregnancy, and even in cases of congestive heart failure in subjects appearing in almost terminal states.

Uremia and Renal Insufficiency.—Organic renal disease, primary or secondary, of such an advanced nature as to cause uremia responds little or not at all to Hyphex. The blood pressure may be moderately lowered without adverse results, the element of renal vasospasm partly combatted, but the progress of the disease is at the most slowed, postponing the inevitable outcome for a few weeks or months. Uremia therefore constitutes one limit of effectiveness for Hyphex, as might be expected, unless the progress of the renal damage is relatively static.

Moderate nitrogen retention (NPN up to 50 or 60 mg. per cent)* may remain when the blood pressure is carefully lowered and after several months may regress, an unexpected phenomenon. Slight nitrogen retention (NPN 26 to 35 mg. per cent) may disappear completely with the establishment of normotension for several months. When the NPN has been consistently much over 60 mg. per cent, a fall is not to be expected (Table 91).

Cerebral Arteriosclerosis and Thrombosis.—It is extremely doubtful that Hyphex prevents the occurrence of thromboses in cerebral arteries severely

* By the method used in this hospital 25 mg. per cent is the upper limit of normal.

T BLE 93 – T XICITY T HEXAMETH NIUM I ON (C)

Pt	Sex	Race	Age	I n B I syst /d t mm Hg	Level of C plasma (g r e l) a l day st n t le				Oral d e C gm /d y	T e on C (u eks)	N P N bl od r g o	Remarks
M S	♀	N	47	~ 5/140	12 (1) 160/90 Ye	11 (3) 150 85 Yes	5 (7) 175/95 No	1 (15) 150 90 No	˷ ˷	25	48	Mal g nant Hype tens on Recovered but died uremia No furtl r tre tn ent
Blood Press u (Syst /D a t m Hg)												
Sym ptom of auto n ic paralys s									5	-	38	M l g ant Hype ter s o R co ered but d ed uremia No fu tl er treatm e t
I C	♀	N	7	230/140	5 (1) 115 80 Ye	10 (2) 1 0/100 Ye	0 5 (5) 25/140 No					
Blo d l res re (Syst /D a t mm Hg)												
Sym pt m									? 5	6	32	Mal g ant Hyperter s o H art l ailure Re o ere l still o Hypl ex
J B	♀	W		40/140	3 (1) 160/90 Yes	0 5 (6) 170/100 No						
Blood Pres ur (Sy t /D a t m Hg)												
Sym pt ms									? 5	2	65	Mal g ant Hype ter ion Mild tox city Recovered but died cor nary occlu ion No furtl er treatment
I M	♀	N	52	210/160	3 3 (1) 170 80 Ye	2 5 ( ) 170/90 Ye	0 75 (5) 00/120 No					
Blo l Pressure (Sy t /D a t m Hg)												
Symptoms												
Normal Th r peutic Le el					1 5	1 0	0 5	0 3	0 2	0 1	0 05	Found in 25 patients on 2 5
Number of P t nt					2	3	7	3	4	?	4	Cm per day or mo e

pressure but without improvement in her mental status. Because she was one of our earlier cases and was critically ill, and before we had as much confidence in and respect for the potency of these drugs as we have learned to have, she was given 25.0 mg. of 1-hydrazinophthalazine intravenously about three hours later. Her blood pressure fell during the subsequent twelve hours to 110 mm. systolic and 70 mm. diastolic, where it remained for three days, rising only when nor-epinephrine was given intravenously. She became oliguric. On the fourth day her blood pressure rose precipitously to very high levels, her NPN had also risen. We hesitated to give her more of these drugs. She died of acute pulmonary edema. At autopsy were found the usual signs of malignant nephrosclerosis, in addition, there were multiple small myocardial infarcts, the result of hypotension.

2. A woman suffering from a major psychosis and hypertension was placed on Hyphex according to the usual routine and her blood pressure controlled at reasonable levels. Placebos were substituted and her blood pressure returned to its previous levels. After several days *both* drugs in full amounts were substituted for the placebos, the initial doses being given at 8.00 A.M. She died suddenly at 8.45 A.M. while taking a shower. No autopsy was obtained.

After these experiences we have not had occasion to give these two drugs together as an initial medication.

Severe Immediate Reactions to Hyphex

Considering the nature of the disease with which we are dealing and the obvious potency of the drugs in altering that disease, it is surprising that severe reactions have been so few. The clinical material was of the worst sort, all of the first hundred patients and many of the remainder showing marked pathological alterations in heart, brain or kidney. The ability of the damaged cardiovascular system to adjust to a lower pressure is remarkable.

Aside from the production of cerebral thrombosis in two individuals with malignant hypertension, cardiovascular accidents have not occurred. Coronary occlusion in patients with old myocardial infarcts has not been precipitated by Hyphex. Nitrogen retention has been worsened by Hyphex when carelessly given, in all cases it has regressed to previous levels when the blood pressure was allowed to rise.

Severe Reactions to Hexamethonium Ion

Hexamethonium Poisoning.—In renal insufficiency it is probable that retention of that portion of hexamethonium ion ingested (10 per cent) or injected (50 per cent) which is normally excreted might be retained. In that event accumulation and poisoning would occur. We have encountered four cases of poisoning and two others which may have suffered from toxicity, but were undiagnosed because we had not developed the method for measuring hexamethonium ion in blood and urine.

TABLE II. OBSTRUCTIVE EFFECTS [illegible] CAUSED BY HEXAMETHONIUM CHLORIDE

No. Cases	Previous Condition	Site	Effect of Hexamethonium [illegible]	Treatment [illegible]	Remarks
1	Intestinal Adhesion [illegible] 10 yrs.	Jejunum	Complete Obstruction	Operation	Recovered
1	Mesenteric Root Syndrome	Duodenum	Partial Obstruction	Intermittent Hexamethonium chloride	Recovered
2	Prostatic Hypertrophy	Urethra	Complete Retention of Urine	Transurethral Prostatectomy	Recovered
2	Prostatic Hypertrophy	Urethra	Partial Retention of Urine	Transurethral Prostatectomy	Recovered
4	Prostatic Hypertrophy	Urethra	Partial Retention of Urine	Urecholine	Recovered
1	Prostatic Hypertrophy	Urethra	Complete Retention of Urine	Catheterization	Recovered
1	Stricture Female	Urethra	Partial Retention of Urine	Catheterization	Recovered
3	Otitis Media old	Eustachian Tube	Serous Otitis Media	Myringotomy	Recovered
6	Otitis Media [illegible]	Eustachian Tube	Serous Otitis Media	Vasoconstrictor	Recovered
1	?	Parotid Gland	Acute Parotitis	Antibiotics	Died
					Unknown

Summary		
	Prostatic Obstruction	11
	Urethral Obstruction	1
	Intestinal Obstruction	2
	Eustachian Tube Obstruction	[illegible]
	Parotid Duct Obstruction	1
	Total	24
	Incidence per cent	[illegible]

The findings are shown in Table 93. Two of these cases may have been complicated by toxicity due to 1-hydrazinophthalazine. All but two occurred in Negroes; nitrogen retention was universally present.

The signs and symptoms of hexamethonium intoxication are those of complete autonomic paralysis and include fixation of the pupil of the eye, dryness of the mouth, dryness of the skin, gastrointestinal atony with ileus, vomiting, and postural hypotension or marked postural fall in blood pressure. Diagnosis can be established by measuring the level of hexamethonium ion in the blood.* Amounts in excess of 1.0 mg. per cent are abnormal; severe symptoms can occur with levels of 2.5 mg. per cent, although we have observed one patient with 16.0 mg. per cent who recovered. Anuria is a serious complication.

Treatment consists of measures to empty the gastrointestinal tract as soon as possible. Enemata are of little avail. Drastic purgatives and urecholine may be necessary. Considerable hexamethonium chloride is retained in the small intestine when toxicity is established; this unabsorbed quantity may continue to be absorbed into the blood for several days after oral medication is discontinued. In one case we have observed the level in the blood rise from 11.5 to 16.0 mg. per cent during the three days after the drug was discontinued but before the bowels had moved. If the blood pressure falls to hypotensive levels continuous intravenous infusion of nor-epinephrine may be necessary.

No deaths from recognized chronic hexamethonium intoxication have occurred. The severe nature of the renal disease, however, which predisposes to retention has made death from uremia inevitable in these cases when Hyphex was discontinued. There is no evidence at present that poisoning hastens the progress of renal failure.

Effect of Hexamethonium Ion on Partial Obstruction of a Hollow Viscus.—When partial organic obstruction of a hollow viscus is present, even if there are no symptoms therefrom, and that viscus is innervated by autonomic nerves, the additional hypotonicity induced by ganglionic blocking agents may result in complete obstruction. Table 94 lists the cases in which complete obstruction of the eustachian tube, parotid duct, duodenum, jejunum and urethra have occurred as a result of the paralytic action of hexamethonium ion. In the small intestine and urethra operation has sometimes been necessary. When Hyphex is mandatory for prolongation of life, transurethral resection of an enlarged but hitherto asymptomatic prostate gland is essential. A history of obstructive symptoms requires caution in administering this agent.

* The method employed is that of Zaimis (British Journal of Pharmacology and Chemotherapy, 5, 424, 1950) modified by H. Mitchell Perry, Jr. For urine the method

then dissolving it completely in a small volume of acetone and reading the acetone at
hexamethonium/ml. of ace-
For blood, where the con
ed a turbidometric method
based on this same hexamethonium reineckate formation.

Immediate Reactions.—This agent acts to inhibit the enzyme which dest ... We first suspected such an ... but nosts ... which slight edema sometimes exhibiting the ... appears when histamine is injected intradermally. Investigation of the *in vitro* action of a number of hydrazines was made at our request and most of them were found to be antihistaminases.[5] The mechanism is not known but may lie in the affinity of hydrazine for metals if histaminase proves to have a metallic coenzyme.

TABLE 96.—LIST OF MINOR SIDE EFFECTS OF 1-HYDRAZINOPHTHALAZINE OBSERVED IN ONE OR MORE PATIENTS

Effect observed	*Usual duration days*	*Treatment*	*Remarks*
Hypertensive headache often prostrating	2-7	Antihistamines	Less apt to occur when hexamethonium ion is given
Generalized slight edema	2-5	None	Disappears spontaneously
Coryza	2-5	None	
Injection of conjunctivae	Many	None	No symptoms
Lassitude and weakness	2-3	None	Recurs for 1 to 3 months
Fever	1-2	None	Disappears spontaneously
Generalized aching, other flu-like symptoms	3-5	Antihistamines	Disappears spontaneously
Prostration	2-3	None	Disappears spontaneously
Palpitation	4-8	None	Disappears spontaneously
Slight anemia	20-60	Ferrous salts	Often disappears without treatment
Diencephalic flush	? 10	None	In neurogenic hypertension
Nausea and vomiting, anorexia	1-3	None	Disappears spontaneously
Tingling and numbness of extremities	—	Reduce dosage	On very large doses only

Many of the immediate reactions caused by the hydrazines can be explained on the basis of excess histamine in the body (Table 96). These are histaminic headaches, flushing, injection of the conjunctivae, stuffiness of the nose, aching of the back, generalized but slight and trans

... aneous. Maintenance of levels of dosage however without increasing them daily may mitigate the severity of the reactions.

The full blown symptomatology of severe reactions with fever resembles an upper respiratory infection similar to mild influenza. We do not know whether the release of excess histamine predisposes the subject to systemic infection with a virus carried in the nasopharynx but previously non-infective or the effects of histamine on the organism mimic that of a virus. The first explanation is more likely because the highest incidence of

Side Effects and Severe Toxic Reactions to 1-Hydrazinophthalazine

It is important to distinguish between the so called side effects of hexamethonium ion, which are actually due to the primary action of the drug itself, and those of 1-hydrazinophthalazine which are true and undesirable side actions. Those due to hexamethonium ion have been discussed in Chapter 19 and are all the result of parasympathetic inhibition.*

Table 99.—Summary of Significant Findings in Acute Fatal Toxicity Due to Hyphex (Probably 1-Hydrazinophthalazine)

Symptom and sign	J J ♂	B J ♂	D M ♂	E M ♀	Remarks
Age	33	49	5[illegible]	52	
Race	N	N	N	N	
Malignant Hypertension	Yes	Yes	Yes	Yes	Three very severe
Nitrogen in blood mg per cent	[illegible]	6[illegible]	40	48	
Heart Failure	Early	No	No	Severe	
Response to Hyphex	Fair	Fair	Fair	Good	
Dose Hexamethonium Gm per day	[illegible] 0	6 0	2 5	[illegible]	Two taking large dose
Dose 1 Hydrazinophthalazine Gm per day	0 9	1 2	0 [illegible]	0 [illegible]	Two taking large dose
Duration of Treatment Months	4	1	[illegible]	5	
Toxic Stage					
Severe Tachypnea	Yes	Yes	Yes	Yes	Worse standing r lieved lying
Duration Days	7			7	To d ath in three
Fever Degrees C	39	3[illegible]	38 4	3	
Venous Pressure mm H_2O	120	17[illegible]	1[illegible]5	108	
Circulation Time Arm to Tongue Sec	20	1[illegible]	13	18	
X Ray Pulmonary Congestion	Yes	Yes	Yes	—	Ed ma pat hy and hilar
Nitrogen in Blood mg per cent	2[illegible]	60	47	36	
Sodium in Serum mEq /l	137	137	128	14	
Carbon dioxide combining power mEq /L	1[illegible]	8	4	[illegible]3 [illegible]	
pH of Blood venous	[illegible]	7 4	—	[illegible] 43	
Urine pH	[illegible]	4 [illegible]	4 [illegible]	4 7	
Cause of Death				Uremi	
Autopsy Findings					
Pulmonary Congestion	Yes	Yes	Yes		
Malignant Sclerosis	No	Yes	Yes	Yes	
Interstitial Pneumonia	Yes	Yes	Yes	Yes	

Note Four
Table 98) died
Two others in s
signs of interstitial p

I n ... o constipation has been the most prevalent side effect of hexa

(
l
to m ... taste perception food and in some cases certain foods have lost

TABLE 37. SEVERE REACTIONS TO 1-HYDRAZINOPHTHALAZINE IN 300 PATIENTS

Pt	Sex	Race	Age	Type of Reaction	Measures Required	Remarks
					Immediate Reactions	
B I	♀	W	45	Angina Pectoris	Discontinuation	Severe Angina before treatment. Maintained on C_6 alone. Calcific Aortic Stenosis
A M	♀	W	62	Angina Pectoris	Discontinuation after fever; intolerant to drug	Maintained on C_6 alone
C W	♀	W	43	Fever 3 times; Prostration, flu	Discontinuation	On Hyphex. No fever third time drug given
M I	♀	W	58	Fever, toxic	Temporary Discontinuation	Maintained on C_6. Severe neurodermatitis previously
E R	♀	W		Severe skin eruption	Discontinuation	Maintained on small doses
D F	♂	W	45	Fever, flu, Prostration	Temporary Discontinuation	Maintained without symptoms
H W	♀	W	41	Fever, flu, Prostration	Substitution of C-6130	Maintained without symptoms
I F	♀	W	33	Fever, flu, Prostration	Substitution of C-6130	
				Reactions Occurring after 6 Months		
K H	♀	W	51	Severe Arthritis, rash	Discontinuation	Recovered
B R	♀	W	43	Severe Arthritis; Fever, Splenomegaly	Discontinuation; Cortisone	Normotensive on C_6 alone. 6130 [illegible] C-7441 caused reaction with arthritis. Recovered
M R	♀	W	48	Severe Arthritis	Same	Normotensive except on Cortisone. Recovered
T I	♂	W	51	Fever, Leucopenia, Splenomegaly, Hematuria, Arthritis	Same	Hyphex begun again with recurrence
A S	♀	W	45	Severe Arthritis, Fever	Discontinuation; Salicylates	Recovered
M D C	♂	W	43	Severe Arthritis, rash	Same	Recovered spontaneously
D T	♂	W	40	Arthralgia, anemia	None	Recovered spontaneously
A W	♀	W	42	Arthralgia	None	Recovered
T S	♀	W	38	Gout	Colchicine	

Summary: Immediate 8 Requiring Discontinuation 4 (1.3 per cent)
Late 9 Requiring Discontinuation 6 (2.[illegible] per cent)

"flu like" reactions coincides with the highest seasonal incidence of respiratory infections

Four individuals of 300 have been unable to take the hydrazines because of repeated (at least three) attacks of high fever, chilly sensations, prostration malaise and headache (Table 97) The explanation for the sensitivity of these individuals is unknown One developed severe dermatitis Seven in all could not take the drug

Late Toxic Reactions—Because the continuous administration of any foreign potent chemical compound to human beings is usually and eventually accompanied by reactions of revulsion sometimes with fatal disturbances, we suspect all untoward events in our patients as being caused by the drug until proven otherwise Diseases and disturbances occurring and coming under suspicion are as follows

1 *Anemia*—A slight degree of secondary anemia has been observed in a fair number of individuals Never have we seen the red blood count below 3 5 million per cu mm or the hemoglobin concentration below 10 Gm per cent The anemia responds readily to the ingestion of ferrous salts

2 *Hepatitis*—One case of what appeared to be infectious hepatitis developed The patient recovered spontaneously although the drug was discontinued The incidence about 0 5 per cent is not unduly high and probably represents chance occurrence

3 *Gout*—One woman who had suffered from the malignant stage of hypertension with moderate nitrogen retention developed gout proven by elevated levels of uric acid in her blood The nitrogen retention had disappeared before the gouty arthritis developed She recovered on the usual measures while still taking Hyphex

4 *Rheumatoid Arthritis* The incidence of rheumatoid arthritis is significantly less in hypertensive than in non hypertensive persons Six individuals taking Hyphex for six months to two years have developed transient arthralgia not requiring treatment Six others developed severe acute migratory or persistent arthritis an incidence of 2 5 per cent Small doses of cortisone controlled the arthritis in two and it did not recur In one patient an acute attack with fever swelling and pain was precipitated five times by 1-hydrazinophthalazine 1-4 dihydrazinophthalazine and 1 hydrazino-4 methyl phthalazine while she was taking placebos and hexamethonium chloride and in two others acute attacks were precipitated by the first of these drugs Therefore in at least three instances severe arthritis was definitely shown to be related to hydrazines It regressed in all cases when the drug was discontinued Two had been in malignant stages of hypertension (Fig 151) A localized skin rash was seen in three The condition was indistinguishable from acute early rheumatoid arthritis Blood pressure had been very well controlled in all cases and continued to be controlled by hexamethonium chloride alone

5 *Disseminated Lupus*—In a fifty five-year-old man suspected but unproven disseminated lupus developed characterized by arthralgia an attack of hepato and splenomegaly fever leucopenia hematuria nitrogen retention and loss of weight He had suffered from three episodes of early

TABLE I. SEVERE REACTIONS TO 1-HYDRAZINOPHTHALAZINE IN 300 PATIENTS

Pt	Sex	Race	Age	Type of Reaction	Measures Required — Immediate Reaction	Remarks
B K	♀	W	45	Angina Pectoris	Discontinuation	Severe Angina before treatment
A M	♀	W	62	Angina Pectoris	Discontinuation for 1 year. Later able to tolerate drug	Maintained on C-6 alone. Calcific Aortic Stenosis
A B	♀	W	43	Fever 3 times. Prostration, flu	Discontinuation	Maintained on C-6 alone
M I	♀	W	58	Fever twice	Temporary Discontinuation	On Hyphex. No fever third time drug given
E R	♀	W	[illegible]	Severe Skin eruption	Discontinuation	Maintained on C-6. Severe neurodermatitis previously
D T	♂	W	48	Fever, flu, Prostration	Temporary Discontinuation	Maintained in small doses
H W	♀	W	41	Fever, flu, Prostration	Substitution of C-6130	Maintained without symptoms
I I	♀	W	33	Fever, flu, Prostration	Substitution of C-6130	Maintained without symptoms
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B R	♀	W	43	Severe Arthritis. Fever, Splenomegaly	Discontinuation. Cortisone	Normotensive on C-6 alone. 6130 & 7411 caused reactions with arthritis
M R	♀	W	45	Severe Arthritis	Same	Recovered
T I	♂	W	[illegible]	Fever, Leucopenia, Splenomegaly, Hematuria, Arthritis	Same	Normotensive except on Cortisone
A S	♀	W	48	Severe Arthritis, Fever	Discontinuation. Salicylates	Recovered. Hyphex begun again with recurrence
M D C	♂	W	49	Severe Arthritis, Rash	Same	Recovered
D T	♂	W	46	Arthralgia, anemia	None	Recovered spontaneously
A W	♀	W	42	Arthralgia	None	Recovered spontaneously
T S	♀	W	48	Gout	Colchicin	Recovered

Summary: Immediate 8. Requiring Discontinuation 4 (1.3 per cent)
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5 *Disseminated Lupus* —In a fifty five year-old man suspected but unproven disseminated lupus developed characterized by arthralgia an attack of hepato- and splenomegaly fever leucopenia hematuria nitrogen retention and loss of weight He had suffered from three episodes of early

LEGEND FOR PLATE II

– in ocular fundi induced by Hyplex

Fundi of T. C. borderline renal

grade 2

NOTE In this and the subsequent plate the dark spots and the faint red spots . . . in the center of the photographs are artifacts inherent in the telescopic camera. All photographs were made by Mr William A. Moor Department of Ophthalmology Washington University School of Medicine through the courtesy of Dr William M James

LEGEND FOR PLATE III

Evolution of changes in ocular fundi induced by Hyplex

First and third rows control second and fourth rows after variable periods of therapy with Hyplex

1 Photograph taken J

from severe malignant

mm Hg. 2 Same eye

disappearance of papilledema hemorrhage and exudates

3 Photograph taken December 22 1951 of the left eye of C. M. a 48 year old woman suffering with malignant hypertension with nitrogen retention and congestive heart failure 4 Same eye on January 11 1952 after 18 days of therapy with grade 0 control The exudate in the center of the photograph has now shrunk to a tiny white spot Papilledema has . . . wared

5 Photograph

with Cushing

taken December

exudates a poor response

C. a 4 year old man with Same eye on May 2 195 aring of papilledema hemorr

rhages and exudates

Photograph taken January 2 1952 of left eye of M. O. C. a 4 year old man

early malignant hypertension moderate nitrogen retention and preeclampsia . . . Same eye on May 21 1952 after . . . weeks of grade 1 control Note the disappearance of papilledema hemorrhages and exudates Response favorable blood pressure . . . Fig 1 8

malignant hypertension, all reversed by hydrazines The condition was controlled by cortisone, at least temporarily A forty-nine year old man with an excellent therapeutic result developed arthralgia anemia, hematuria, albuminuria, elevated NPN and signs of liver damage in two months he has partially recovered spontaneously while still taking small doses of hydrazine No L-E' cells were found in the blood or bone marrow of either patient The resemblance of the syndrome to disseminated lupus erythematosus is obvious

6 *Interstitial Pneumonia* —A fifty four-year-old man who had suffered from congestive heart failure and malignant hypertension reversed by Hyphex died within three days of interstitial pneumonia after receiving the drugs for two months It is not known which if either drug was implicated

A similar condition has occurred in three Negro men and one Negro woman Severe tachypnea most marked in the upright position is the characteristic symptom respiratory rates as high as 90 per minute have been observed The patient usually feels better recumbent The respiration is shallow The tachypnea is not at first incapacitating Signs of pulmonary congestion are minimal to absent although *x* ray photographs show severe congestion spotted throughout the lungs or spreading from the hilar regions Signs of cardiac failure (prolonged circulation time elevated venous pressure) are absent or minimal (Table 9b) Acute interstitial (fibrotic) pneumonia was found at autopsy

Laboratory examination is of little aid in arriving at an explanation for the symptoms except that the pH of the urine has been invariably acid and the pH of the blood alkaline Three individuals have had elevated levels of hexamethonium ion in blood one did not The condition was fatal in the three men the woman recovered but later died of uremia

We are at a loss to explain this circumstance All patients suffered from the malignant stage three exhibited renal insufficiency There are two current explanations one physical the other chemical It is possible that hexamethonium ion in large amounts blocks normal neurogenic vasomotor tone in the pulmonary vascular bed[4] without interfering with reflexes from the lungs to the respiratory center If this were so and cardiac output remained normal pressure would be transferred from pulmonary arteries and arterioles to capillaries causing forward congestion This congestion might set in motion the dyspneic reflex and fibrosis of the lungs

The second chemical explanation involves the affinity of 1 hydrazinophthalazine for metals Zinc is a necessary co-enzyme for carbonic anhydrase removal of zinc would be expected to interfere with transport of carbon dioxide across the pulmonary alveolar membrane and with the excretion of bicarbonate by the kidney Hydrazines inhibit histaminase for example If the tachypnea were due to accumulation of carbonic acid in the blood one would expect to find the carbon dioxide content of arterial blood elevated Unfortunately content has not been measured combining power has varied from low to slightly high values Concentrations of sodium have not been abnormal The affinity of hydrazine for zinc how

Legend for Plate II

Fund. II F. C.

grade 2

Note. In this and the subsequent plate the dark spots and the faint reflections in the center of the photographs are artifacts inherent in the ... scopic camera. All photographs were made by Mr William A. Moor, Department of Ophthalmology, Washington University School of Medicine, through the courtesy of Dr William M James.

Legend for Plate III

Evolution of changes in ocular fundi induced by Hyplex

First and third rows ... control, second and fourth rows after variable period of therapy with Hyplex

1 Photograph taken January 12 1952 of left eye of J. C., a 34 year old man suffering from severe malignant hypertension whose diastolic pressure ranged from 170 to 180 ... 2 Same eye on May 21 1952 after 5 months of grade 0 control. Note the ...

h..p

3 ... M. a 48 year old woman ... and congestive heart

uffer ... rapy with grade 0 control

Exudate ...

The exudate in the center of the photograph has now shrunk to a tiny white spot

Papilledema has di...

5 Photograph t...

with Cushing's sy...

... December 3 ...

... exudates, a poor response

C., a 47 year old man with ... Same eye on May 27 1952 ... rang... of papilledema ...

... stages and exudates

7 Photograph taken January ...

suffering from ... malignant

week of Hyplex therapy and gr...

... us appears to be ...

... 9 ... taken December ... left eye of T. ..., a 3 year ... woman with ... malignant hypertension ... moderate nitrogen retention and hypercholesterolemia ... 10 Same eye on May 21 1952 after ... weeks of grade ... control. Note the disappearance of papilledema, hemorrhages and exudates. Response of average blood pressure ... shown in Fig. 18

PLATE II


1

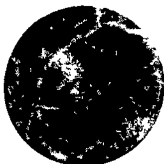

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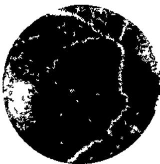

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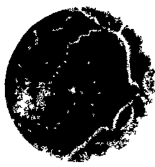

12

PLATE III


1


3


5


2


4


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7


9


11


8


10


12

TABLE 98.—CAUSE OF DEATH IN PATIENTS ON HYPHEX

Pt	Sex	Race	Age	Therapeutic grade	Stage of hypertension	Complicating factors	Cause of death	Remarks
W G	♂	W	62	2	IVc	Cerebral Hemorrhage, Renal Insufficiency	Parotitis and Pneumonia	B P well controlled
								Normotensive
A K	♂	W	55	2	IVb	Congestive Heart Failure	Interstitial Pneumonia	B P well controlled
C M	♀	W	48	1	IVc	Congestive Heart Failure, Renal Insufficiency	Died in sleep	B I well controlled
								Normotensive
E M	♀	W	52	1	IVc	Congestive Heart Failure, Renal Insufficiency	Died in sleep	B P well controlled
								Normotensive
A G	♀	N	29	3	IVb	Cerebral Edema 3 times	Post operative shock	Sympathectomy because of low intelligence
L K *	♀	W	32	4	IVc	Encephalopathy Coma, Renal Insufficiency	Pulmonary Edema	B I became tolerant to Hyphex
H A	♂	W	57	3	III	Arteriosclerosis Severe, Sympathectomy	Cerebral Accident (?Thrombosis)	B P partly controlled
H F	♂	W	52	1	IVa	Congestive Heart Failure	3 Cerebral Thromboses	B P well controlled
M S	♀	N	48	3	IVc	Uremia	Uremia	B P fair control
W J †	♂	N	49	4	IVc	Renal Insufficiency	Interstitial pneumonia	B P poor control
D M †	♂	N	55	4	IVc	Renal Insufficiency	Interstitial pneumonia	B I fair control
J J †	♂	N	33	3	IVa	Early Congestive Heart Failure	Interstitial pneumonia	B I fair control
C F	♀	W	13	1	IVc	Uremia Encephalopathy	Uremia	B P well controlled
H K	♂	W	37	1	III	Congestive Heart Failure	Sudden Death	B P well controlled
C C	♂	W	46	4	IVc	Congestive Heart Failure, Uremia	Uremia	B P fair control
C S	♂	W	49	1	IVa	Coronary Occlusion	Cancer of Lung	B I well controlled
F K	♂	W	53	3	IVc	Uremia	Uremia	B P fair control

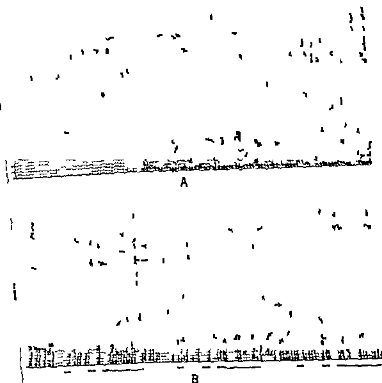
* See Fig 160
† See Table 9[illegible]

Summary

Cause	Number
Uremia	4
Pulmonary Edema	1
Cerebral Thrombosis	2
Sudden Death Cause Unknown	3
Pneumonia	1
Post-operative Shock	1
Interstitial pneumonia	4
Cancer	1

related to the hypertensive process or was due to uremia already established when treatment was begun. These individuals were all very ill at the time of treatment. Toxicity from the drugs caused … pneumonia 1 … shock 1 uremia 4 cerebral arterial thrombosis 1, cancer of … ses from unknown causes … Only 1 discussed below, …

… sulting from hypertensive … gues… at the cause in the three who …



Blood pressure was controlled in hospital at reasonable (Grade 2) levels in February 1952 (upper graph). During the next eight months she became normotensive but developed severe arthritis which was made worse by each of three hydrazines (5968 … 441). Readmission for arthritis is shown in the lower graph (January 1953). … had been substituted for the hydrazines but in hospital each drug was cau… tiously given for one day only. The two "spikes" of blood pressure occurred during

TABLE 99.—EFFECT OF HYPHEX ON CONGESTIVE HEART FAILURE DUE TO HYPERTENSION

Patient	*Sex*	*Race*	*Age*	*Stage of hypertension*	*Previous functional class # and time (mos.)*	*Previous cardiac and diuretic drugs used*	*Result of Hyphex*	*Duration of compensation (mos.)*	*Low salt diet used*	*Other drugs necessary*	*Functional class # during treatment*
I M	♀	N	47	Malignant	IV 24	Dig Hg Low Salt	Excellent	18	No	None	I
J B	♀	W	42	Malignant	IV 3	Dig Hg Low Salt	Excellent	10	No	None	II
D B	♂	W	42	Benign	IV 12	Dig Hg Low Salt	Excellent	12	No	None	I
H F	♂	W	32	Malignant	III 3	Dig Hg Low Salt	Excellent	10*	No	None	I
H H	♂	W	42	Malignant Renal Insufficiency	IV 12	Hg Low Salt	Excellent	10	No	None	II
A K	♂	W	55	Malignant	IV 24	Dig Hg Low Salt	Good	3**	Yes	None	II
J H	♂	W	38	Malignant	IV 1	Dig Hg Low Salt	Good	9	No	None	I
H J	♂	W	55	Benign	IV 3	Dig Hg Low Salt	Excellent	9	No	None	I
H K	♂	W	37	Benign Severe	III 12	Dig Hg Low Salt	Excellent	3†	No	Dig	I
N K	♂	W	58	Benign Arteriosclerotic H D	IV 6	Dig Hg Low Salt	Excellent	13	No	None	II
I M	♀	N	52	Malignant Renal Insufficiency	IV 3	Dig Hg Low Salt	Good	3‡	No	None	II

C M	♀	W	5	Malignant Renal Insufficiency	IV 10	Dig Hg Low Salt	Good but ur...e	1††	No	No	II
R S	♂	W	5	Benign Arteriosclerotic H D	IV 1	Dig Hg Rice	Good less failure	10	Y	Dig Hg	II
S S	♀	W	56	Benign Arteriosclerotic H D Aur Fib	IV 1	Dig Hg Low Salt	Poor Uncooperative	—	Yes	Dig Hg	III
M Z	♀	W	3	Benign Severe Mitral Stenosis	I 0	None	Developed Failure 1 Year	—	Yes	Dig Hg	III
J J	♂	W	6	Benign Arteriosclerotic H D	IV 12	Dig Hg Low Salt	Excellent	2	No	None	I
G G	♂	W	41	Malignant Uremia	IV 1	None	Excellent	8‡‡	No	None	I
C M	♀	W	48	Malignant	IV 5	Dig Hg Low Salt	Excellent	1††	No	None	II

Summary		
	Living	11
	Dead	7
	Died of New Hypertensive Complication	1
	Died of Disorder existing before Hyphex was started	3
	Died of New Intercurrent Disease	1
	Unknown	2

Functional Class — American Heart Association Classification
* Died of Cerebral Thrombosis
** Died of Interstitial Pneumonia
† Died sudden death while at work
‡ Died Coronary Occlusion after discontinuation in hospital
†† Died in sleep without failure
‡‡ Died Uremia

TABLE 99.—EFFECT OF HYPHEX ON CONGESTIVE HEART FAILURE DUE TO HYPERTENSION

Patient	*Sex*	*Race*	*Age*	*Stage of hypertension*	*Previous functional class # and time (mos.)*	*Previous cardiac and diuretic drugs used*	*Result of Hyphex*	*Duration of compensation (mos.)*	*Low salt diet used*	*Other drugs necessary*	*Functional class # during treatment*
I M	♀	N	47	Malignant	IV 24	Dig Hg Low Salt	Excellent	18	No	None	I
J B	♀	W	42	Malignant	IV 3	Dig Hg Low Salt	Excellent	10	No	None	II
D B	♂	W	42	Benign	IV 12	Dig Hg Low Salt	Excellent	12	No	None	I
H F	♂	W	52	Malignant	III 3	Dig Hg Low Salt	Excellent	10*	No	None	I
H H	♂	W	42	Malignant Renal Insufficiency	IV 12	Hg Low Salt	Excellent	10	No	None	II
A K	♂	W	55	Malignant	IV 24	Dig Hg Low Salt	Good	3**	Yes	None	II
J H	♂	W	58	Malignant	IV 1	Dig Hg Low Salt	Good	9	No	None	I
H J	♂	W	55	Benign	IV 3	Dig Hg Low Salt	Excellent	9	No	None	I
H K	♂	W	47	Benign Severe	III 12	Dig Hg Low Salt	Excellent	3†	No	Dig	I
N K	♂	W	58	Benign Arteriosclerotic H D	IV 6	Dig Hg Low Salt	Excellent	13	No	None	II
I M	♀	N	52	Malignant Renal Insufficiency	IV 3	Dig Hg Low Salt	Good	5‡	No	None	II

became more severe and attacks more frequent therefore she took only hexamethonium chloride with partial intermittent control of her blood pressure A second developed recurrence of angina pectoris on the hydra zines and both drugs were discontinued One man was controlled only partly although angina was lessened he became di couraged and di con-

TABLE 100 —EFFECT OF HYPHEX ON ANGINA PECTORIS IN BENIGN HYPERTENSION

Pt	S	Race	Ag	Pathological Condition	Symptoms	Effect on Angina	Remarks
I D	♂	W	50	Coronary Arteriosclerosis	Angina Pectoris Severe	Partial Relief	Small doses of Hyphex only Discontinued drugs after 3 months
B I	♀	W	45	Coronary Arteriosclerosis	Angina Pectoris Severe	Worse initially	Apresoline caused angina Hyphex discontinued
A M	♀	W	62	Calcific Aortic Stenosis	Angina Pectoris, Severe	Complete Relief	Apresoline ceased Hexamethonium alone used
M C	♀	W	50	Coronary Arteriosclerosis	Angina Pectoris Severe	Almost complete relief	Nitrites reduced from [illegible] per day to 6 per month
R S	♂	W	55	Coronary Arteriosclerosis Congestive Heart Failure	Angina Pectoris Moderate	Complete relief	
C S	♂	W	55	Coronary Occlusion Old	Angina Pectoris Moderate	Complete relief	No nitrites necessary
J I	♂	W	48	Coronary Arteriosclerosis	Angina Pectoris Mild	Complete relief	
C B	[illegible]	W	42	Coronary Arteriosclerosis	Angina Pectoris Moderate	Complete relief	Main Depressive Psychosis Discontinued after 3 months
N K	[illegible]	W	55	Congestive Heart Failure Coronary Arteriosclerosis	Angina Pectoris Moderate	Complete relief	No nitrites necessary

the capillaries from too much engorgement On the basis of this one experience we use Hyphex most cautiously in patients with mitral stenosis

Old Coronary Occlusion and Coronary Arteriosclerosis

s with history or electro- Of 35 patients so treated none have developed further symptoms or changes in the electrocardiogram In fact alterations in the direction of normal have occurred in strain patterns

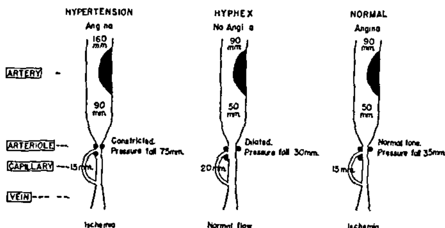


Fig 1 2 —Theoretical explanation of relief of angina pectoris by Hyphex An hypothetical plaque in a coronary artery is shown by the black area which results in a fall of

due to neurogenic and humoral vasoconstrictor substances causes ischemia In the middle is shown relief of ischemia at normotensive levels due to dilatation of the arteriolar bed even though arterial pressure distal to the obstruction is low On the right is shown the same situation with arterioles constricted causing ischemia No one knows whether or not arterioles distal to an obstruction are more sensitive than normal ones to vasoconstrictor influences but perhaps they are All of these values are exaggerated in order to illustrate the point Under other hemodynamic circumstances angina could be produced

One adverse result has been observed a forty-three-year old man with a history of at least two severe episodes of coronary occlusion congestive heart failure and a markedly abnormal electrocardiogram was placed upon Hyphex and his blood pressure controlled Three months later while driving a tractor he fell dead No autopsy was obtained but his death was presumably of cardiac origin

Angina Pectoris —Nine individuals suffered from angina pectoris severe enough to require nitrites for relief One woman exhibited calcific aortic stenosis when 1 hydrazinophthalazine was given on three occasions pain

retina may be quickly relieved (2 cases) blood pressure lowered rapidly and left low (4 cases) but reversal of the process may not occur (1 case) One patient became pregnant while on Hyphex she aborted during the sixth month No live babies have been born from women given Hyphex but adequate experience has not accumulated for definitive answers to be given as to whether or not these drugs are toxic to the developing embryo Hexamethonium ion is said not to be toxic in this particular respect

Comment

Reversal of the severe secondary changes when blood pressure was lowered has been a most encouraging sign We cannot subscribe to the belief that atherosclerosis itself is altered by Hyphex although some of the late results suggest that explanation On the other hand the progress of atherosclerosis may be delayed The severe physical effects of high intra arterial tension are rapidly altered the exudative phase reversed the renal disea normal and the progres s can be determined ld have come as a surprise to us trained and habituated in the harsh pre-treatment school They go to show that the physical and chemical effects of arterial hyperten ion cause most of the damage when properly neutralized dam age ceases and repair begins It will take many years however properly to evaluate the effects of control of hypertension upon life expectancy the short term (two years) is bright with hope

RESULTS IN BENIGN HYPERTENSION

General —In over 200 cases treated or seen by us the blood pressure fell to normotensive reasonable or slightly hypertensive levels and remained there as long as Hyphex was continued Symptoms completely disappeared and all patients felt well after a year or more on therapy In many instances doses were reduced that of hexamethonium chloride by the self-eliminating schedule followed that of 1 hydrazinophthalzaine by the patients of necessity or on trial Secondary pathological changes altered in the direction of normal except when of such a nature as to be obviously irreversible they include loss of albuminuria improvement in renal function slow disappearance of the electrocardiographic and x ray signs of left ventricular enlargement improvement in the appearance of r dia holes

Blood Pressure —Blood pressure taken 3 times a day tends to fluctuate much more widely than is optimal for normotensive persons The morning reading is almost always higher after eight hours without drugs than tho e during the day Usual fluctuations in a well-controlled individual may amount to 40 mm or more systolic (160 to 110) and 20 mm diastolic (90 to 70) although gradual evening of the levels occurs in the long term

y or almost completely (Table 100) Apparently vasodilatation of the arterioles distal to a diseased coronary artery was sufficient to provide adequate flow to capillaries (Fig 152) in some and was insufficient in others

Old Cerebral Accidents

Hyphex was given to 36 individuals who had suffered either a major cerebral hemorrhage or attacks of transient "cerebral angiopathy" No further attacks occurred in 35, one patient already described, suffered a second minor episode involving the same side of her body No cerebral hemorrhages have occurred in any patients on Hyphex Seven cerebral thromboses on the other hand have taken place, four in individuals who had exhibited the malignant phase While it is likely that control of blood pressure may postpone the onset of hemorrhage it is probable that thrombosis of a cerebral vessel may not be prevented and may even be initiated The mental conditions of 12 exhibiting signs of atherosclerotic cerebral softening did not become worse

Nitrogen Retention

Slight nitrogen retention may disappear after the blood pressure is controlled for several weeks or months (Table 91) We have observed this occurrence fourteen times When treated carefully severe nitrogen retention especially that of relatively recent origin may regress somewhat in 5 cases when the NPN was 60 mg per cent or more it fell to much less elevated levels Two of these were in children in the malignant stage Renal vasospasm is probably released by Hyphex the contribution of vasospasm to renal insufficiency can thereby be mitigated without affecting the organic increment *

Toxemia of Pregnancy

The toxemias of pregnancy arise in so far as is known from two disturbances retention of salt and renal damage due to pyelonephritis or other renal diseases The results of Hyphex in toxemias have been inconsistent In 1 case an obese Negro woman exhibiting the endocrine hypertensive syndrome the results were very poor A month in hospital with vigorous treatment failed to lower blood pressure significantly On the other hand a woman who had experienced her third attack of toxemia with convulsions was completely relieved and developed normotension on Hyphex Experience with 7 cases has indicated that detachment of the

* It is entirely possible however that organic renal vascular disease may slowly reverse itself when hypertension is controlled For example we have observed the 15 minute excretion of phenol red in a man with malignant hypertension to increase from 7 5 per cent to 10 per cent in three weeks 15 per cent in five months and 30 per cent in fifteen months Many other examples of return of renal function have been seen slowly progressing

Comment

Reversal of the severe secondary changes when blood pressure was lowered has been a most encouraging sign We cannot subscribe to the belief that atherosclerosis itself is altered by Hyphex although some of the late results suggest that explanation On the other hand the progress of atherosclerosis may be delayed The severe physical effects of high intra arterial tension are rapidly altered the exudative phase reversed the renal disease apparently changing slightly in the direction of normal and the progress of the hypertensive process halted in so far as can be determined Many of these findings were not wholly expected and have come as a surprise to us trained and habituated in the harsh pre-treatment school They go to show that the physical and chemical effects of arterial hypertension cause most of the damage when properly neutralized damage ceases and repair begins It will take many years however properly to evaluate the effects of control of hypertension upon life expectancy the short term (two years) is bright with hope

RESULTS IN BENIGN HYPERTENSION

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much more widely than is optimal for normotensive persons The morning reading is almost always higher after eight hours without drug than those during the day Usual fluctuations in a well-controlled individual may amount to 40 mm or more systolic (160 to 110) and 20 mm diastolic (90 to 70) although gradual evening of the levels occurs in the long term

Emotional "spikes" of blood pressure are modified but not abolished. We first became aware of this fact while treating a woman with early malignant hypertension in hospital. She had an *inordinate* attachment to her only son. On three days of fourteen her systolic pressure rose on a single determination to about 180 mm. and then fell to normal levels, previously it had been controlled. Reconstruction of emotional events revealed that on the first occasion she received word that her son was told to report to his draft board for examination, on the second he called to say that he had

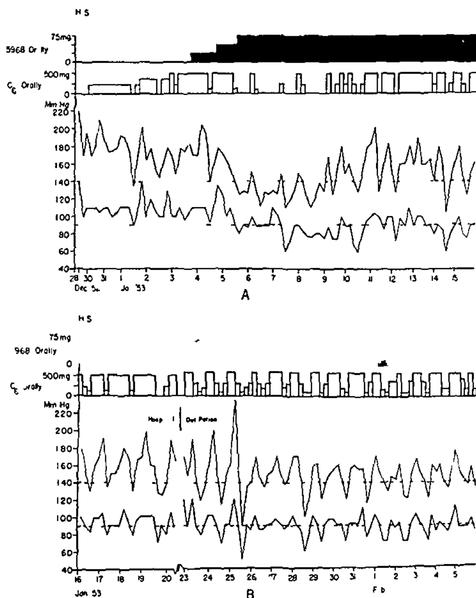


FIG. 153.—Hospital and out-patient charts of blood pressure of H. S., a fifty-eight year-old woman suffering from severe benign hypertension who had been taking hexamethonium chloride for three months with the effect shown. When 1-hydrazinophthalazine was added the blood pressure became lower although it was always elevated at 6:00 A.M. due probably to elimination of hexamethonium ion during the night. Gradually this fluctuation diminished. All readings are shown.

camp An extreme example ... his selling the first three days of the week worked in his office the ... two and stayed at home on week ends For over a year the same pattern was observed levels occasionally up to 180 and 190 mm systolic and 100 to 120 diastolic on Monday Tuesday and Wednesday reasonable or normotensive levels Thursday and Friday and low normotensive levels without ... Saturday and Sunday (taking Hyphex on week ends ... ation he was usually ... Many other hypote ...

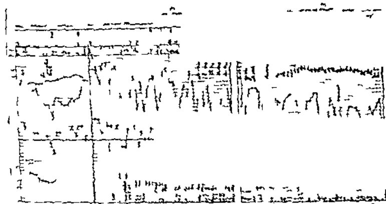


Fig 154 Hospital record of blood pressure of M O C forty-eight who suffer ... usually and he was symptom free a year later it was normal The heavy horizontal line in the middle of the chart represents 150 mm Hg and the top 250 mm

examples of emotional "spikes" have been observed intelligent patients learn to recognize them and avoid the reaction* (Fig 153)

Renal Function —Control of hypertension was always associated with disappearance of albuminuria and improvement in the ability of the kidney to excrete injected phenol red (P S P) Organic renal parenchymal diseases, especially pyelonephritis, remained, several infections were seen to recur without alteration of the level of blood pressure Treatment of renal infections is difficult but must be accomplished to prevent progressive renal damage

Electrocardiographic and X-Ray Signs of Left Ventricular Strain —Alterations toward normal occurred in many cases showing cardiac enlargement and signs of left ventricular strain The change was slow, as a rule al though in many instances improvement in the electrocardiogram appeared within two weeks of the establishment of normotension Lesions such as bundle branch block, old myocardial infarction and the like did not change the ischemic pattern however often disappeared X ray evidence of cardiac enlargement, however was very slow to regress usually requiring six months or more

Ocular Fundi —Grade II ocular fundi usually reverted to grade I and grade I to normal The relief of spasm was striking Tortuous vessels were observed to straighten corkscrew shaped ones to become tortuous The appearance of the fundi did not alter in patients whose control was poor or who discontinued the drugs

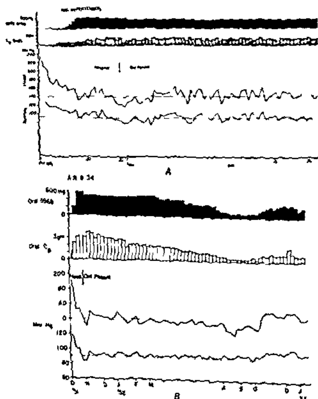
Symptomatic Improvement —Headache was completely relieved in all cases when present Anxiety nervousness tension palpitation emotional perspiration were lessened or abolished The diencephalic flush did not disappear however but attacks were less frequent in patients with neurogenic hypertension After a year on treatment all patients were completely satisfied and symptom free

Resistant Cases —No true and complete resistance to Hyphex was encountered There have been 12 men and 8 women however suffering from benign stages and atherosclerosis aged between forty five and sixty five who appeared partly resistant for several months showing systolic pressures as sporadically high as 200 mm varying to levels as low as 140 mm and averaging about 170 to 180 mm (Therapeutic Grade 3) We cannot account for this variation in results it is the exception which has become of most interest to us Perhaps other factors were operating in them to maintain hypertension (Table 101) It is curious that 14 have suffered from cerebral vascular accidents Eight other individuals appeared partly resistant for three to six months and later became much more normotensive Resistance of this nature was seen in cooperative patients in malignant stages only in four individuals all severely ill and in 6 who had suffered attacks of coma due to cerebral edema even in them significant (more than 30 mm diastolic) and sustained falls in blood pressure occurred Since the incidence of partial resistance was high in those who exhibited cerebral disease it becomes evident that all of the mechanisms of hyper

* In such individuals the use of extracts of *Rauwolfia serpentina* (Serpasil) has served to modify or eliminate these extreme emotional fluctuations

tension are not being neutralized and that special circumstances attend and accompany cerebral vascular disorders

Severe Renal Diseases.—Hyphex has been given cautiously to patients with uremia due to disseminated lupus erythematosus, chronic glomerular nephritis, chronic pyelonephritis, polyarteritis nodosa and the like. Elevated blood pressure was lowered without alteration in the basic disease although secondary effects of hypertension on brain and other organs was often alleviated. All uremic patients die more comfortably in some cases but without obvious prolongation of life



Unsuccessful Sympathectomy —Twenty patients previously subjected to lumbodorsal sympathectomy have returned with hypertension in severe (11 cases) or malignant (9 cases) stages While all responded satisfactorily to Hyphex caution was necessary in avoiding too large doses of hexamethonium ion, for severe postural hypotension could be readily produced

TABLE 101 —SUMMARY OF FINDINGS COMMON TO PARTLY RESISTANT CASES (THERAPEUTIC GRADES 3 AND 4)

Summary	No cases		Living grade 4	Living grade 3	Dead* grade 4	Dead* grade 3	
Malignant Hypertension	89						
With Nitrogen Retention		40		4	4	2	
Severe		29		4		1	
Early		20		2		1	
Cerebral Edema		(8)			(2)	(1)	
Benign Hypertension Severe	139			1		1	
Cerebral Vascular Disease		35		(14)		(1)	
Coronary Arterial Disease		35		(3)			
Total Severe Hypertension	228						
Total Partly Resistant			0	27	4	4	35
Total with Cerebral Disorder				14	2	2	18
Reduction of Average Blood Pressure mm Hg							
Diastolic reduced 10–20				5			
21–30				9	1	1	
31–40				2		2	
41–60				1	1	1	
More than 60					2		
Systolic reduced 30–40				6			
41–50				2		1	
51–60				5	1	1	
61–80				3	1	1	
More than 80				1	2	1	
Uncooperative or unreliable				10*			
Total				27	4	4	

* Including those among the 11 discontinued cases

** Diastolic pressure reduced more than 20 mm when patient cooperates

Both hexamethonium ion and 1-hydrazinophthalazine markedly enhance the postural fall of blood pressure in sympathectomized subjects We have seen systolic changes of 20 to 30 mm converted to ones of 80 or 100 mm especially when the drugs were first administered Therefore a compromise was made in order to provide normal or slightly subnormal levels in the standing position Strange to say half of these patients have achieved Therapeutic Grades of 1 or 0 *in the supine position* after a year or more on Hyphex Surely the hypertensive process has been partly reversed in some of them

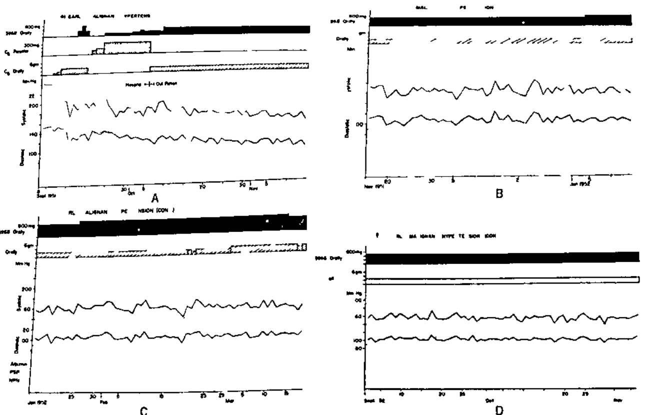
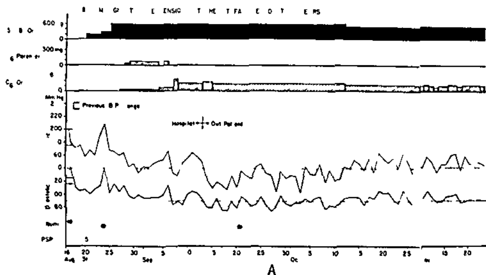
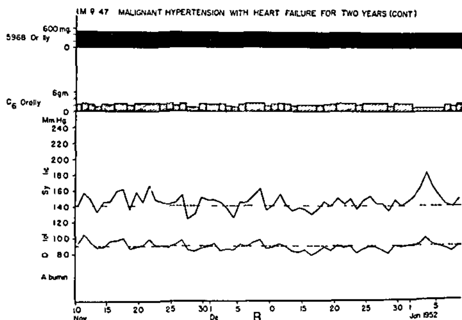


Fig 101 —Resistance to Hyplex slowly disappearing. H C was a forty two-year-old woman with early malignant hypertension and a very high blood pressure which in her physician's office was often 200 to 280 mm systolic and 150 to 170 mm diastolic. She was incapacitated and deteriorating rapidly. It was quite resistant in hospital to both parenteral and oral medication. Continued use however resulted in a slow decline. She works twelve to fourteen hours a day in her own restaurant and is symptom free. At present the therapeutic result is Grade 2



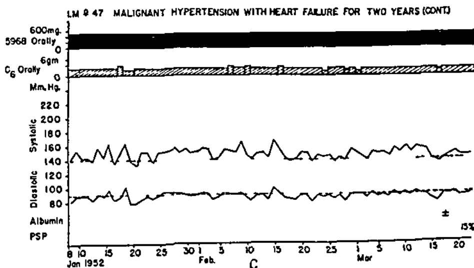
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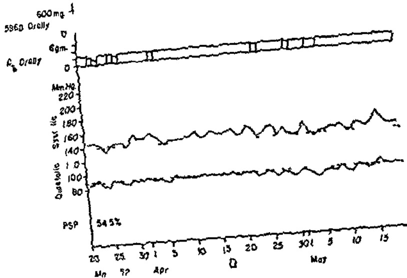
LM 9 47 MALIGNANT HYPERTENSION WITH HEART FAILURE FOR TWO YEARS (CONT)
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5968 Orally
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Mm Hg
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140
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100
80
Albumin
10
Nov
15
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25
30
1
5
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15
20
25
30
1
5
Jan 1952

B



LM 9 47 MALIGNANT HYPERTENSION WITH HEART FAILURE FOR TWO YEARS (CONT)
600mg.
5968 Orally
0
6gm
C6 Orally
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Mm. Hg.
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140
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maalies were to be noted without car ful and exhau tive study She has returned from a desperate almost terminal situation to a normal life Her therapeutic Grade is now D

Deaths from cerebral thrombosis occurred in one severely arteriosclerotic man; a young woman suffered cerebral thrombosis and a uremic man died of uremia, indicating again the limits of the method in failing to reverse irreversible vascular and renal damage.

BLOOD PRESSURE

Since we are treating an elevated blood pressure and thereby attempting to delay or halt the pathological progress of effects secondary to hypertension, in the final analysis a change in the level of blood pressure produced without causing detriment to heart, blood or kidneys is the criterion of effectiveness of the method. In evaluating the results due allowance must be made for psychogenic influences as well as adverse effects upon the bodily economy as a whole. Subjected to the utmost degree of criticism and to the most carefully controlled observations, however, no doubt whatsoever arises that the method is effective.

Method of Analysis of Results

Stage of the Disease.—We have adopted the various stages of hypertensive disease listed in Chapter 18 in order to compare alterations occurring on Hyphex. In addition, we have set up arbitrary standards of blood pressure during treatment periods in an attempt to show changes caused by Hyphex. These standards, which we will call *Therapeutic Grades*, are as follows:

Therapeutic Grades

0 Blood pressure always normal: systolic 140 mm. Hg or below, diastolic 90 or below.

1 Normal level of blood pressure 80 per cent of time: systolic never over 160 mm., diastolic never over 100 mm.

2 Reasonable levels of blood pressure: Systolic 160 mm. or below 80 per cent of time, never over 180 mm.; diastolic 95 mm. or below 80 per cent of time, never over 110 mm.

3 Moderately hypertensive levels of blood pressure: Systolic 180 or below 80 per cent of time, never over 200 mm.; diastolic 100 or below 80 per cent of time, never over 120 mm.

4 Consistently hypertensive levels of blood pressure: Systolic 200 mm. or below 80 per cent of time, diastolic 120 or below 80 per cent of time.

Since Hyphex always causes reductions of blood pressure even in the most severe cases, no lesser therapeutic grade is necessary.

Control Stages (see Table 3)

0 Prehypertension: Systolic usually 140 mm. or below, diastolic usually 90 mm. or below except under stress.

I Mild Benign: Normal at complete rest; systolic 150 to 180 mm. most of time, diastolic 90 to 105 mm. most of time.

II Moderate Benign Normal only under heavy sedation during sleep Systolic 180 to 220 mm most of time diastolic 105 to 120 mm most of time even at rest

III Severe Benign Never normal Systolic 200 to 270 mm all of time diastolic 120 to 160 mm all of time

IVa Malignant—Early Systolic 200 to 280 mm all of time diastolic 130 to 160 mm most of time Fixed diastolic pressure

IVb Malignant—Severe Systolic 200 to 300 mm all of time diastolic 150 to 200 mm all of time

IVc Malignant—Decompensated Systolic 200 to 300 mm all of time diastolic 130 to 220 most of time unless in heart failure Nitrogen retention present

TABLE 102 —EFFECT OF HYTHEX ON BLOOD PRESSURE

(PATIENTS ON TREATMENT 6 TO 24 MONTHS)

Therapeutic grade	4	3	2	1	0	Total
Living						
Initial Stage						
IVc (N Retention)		–	11	3	1	22 (4)
IVb (Severe Malignant)		5	12	6	2	2[illegible] (3)
IVa (Early Malignant)		[illegible]	8	2	1	1[illegible]
III		26	63	2[illegible]	4	122
II			11	[illegible]	4	20
	0	43	105	45	12	205
Dead						
IVc	4	2	1	3		10
IVb		1	1			2
IVa		1		2		3
III		1		1		2
	4	5	[illegible]	6		17

Note

... in ... p ... except those marked in parentheses

All blood pressure determinations discussed in this chapter were taken by nurses occasionally checked by physicians We have compared nurses readings in hospital with patients readings at home more nearly comparable sets of figures are thereby obtained than by comparing physicians readings in hospital (which are usually higher than nurses) with patients at home (the results would appear better than they are) or physicians pressures in hospital with physicians pressures at home (the results would appear better or worse than they are depend...

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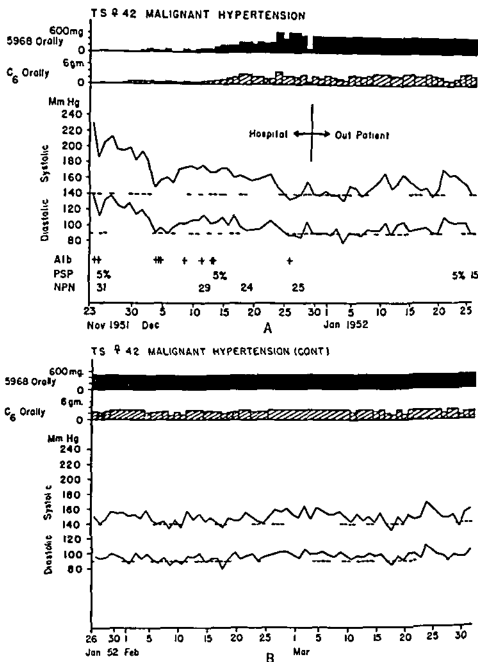


Fig 158 —Record of T S a forty two-year old woman with malignant hypertension and slight nitrogen retention on the basis of chronic pyelonephritis Ocular fundi were grade IV Blood pressure and the malignant stage were easily controlled She was re-admitted to hospital in May 1952 when it was found that her renal function had im

chloride (C)6 Fundi shown in Color Plate III

control levels and the nearest highest
variations are great from moment to moment

In Table 102 are seen the changes from the original stage of hypertension to a new therapeutic grade Reversal of the secondary effects of hypertension are not considered in this analysis It will be noticed that a majority whatever the original stage or level of blood pressure have reverted to Grades 2 and 1 A few have become normotensive and some are partly resistant (Figs 154-158)

Effect of Administration of Placebos —It was found impossible to give placebos to patients suffering from malignant hypertension the result was too hazardous to the health of the patient In the few times they were given blood pressure became alarmingly high within eight to twelve hours one patient developed emi-coma during ten hours It was likewise impossible to give placebos to patients suffering from severe benign stages

of therapy when blood pressure may not rise for several
continuation could placebos be substituted for the actual drugs Because the method is so effective control studies of this nature were limited by the immediate awareness of the patient that something had gone wrong We were able however to administer them for several weeks to one individual who developed severe arthritis in order to observe the effect upon symptoms of 1 hydrazinophthalazine she had become practically normotensive without needing the drug (Fig 151) It is of course unnecessary and unwise to substitute placebos for penicillin or insulin

Control Periods —In all cases control levels of blood pressure were established
in bed in ho
four hours
being administered the variations in blood pressure served as a sort of control No case was accepted for treatment if blood pressure fell to normal

the
like They are not usually necessary Occasionally we have used Mephenesin for anxiety and nervousness Low-salt diets are contraindicated in nitrogen retention for Hyphex may enhance the salt losing tendency of damaged kidneys causing the low-salt syndrome We have observed the low salt syndrome appear 3 times in 1 patient with nitrogen retention while on a normal intake of sodium chloride Undoubtedly the hypo-

to control edema The only common restriction is that of tobacco for nicotine acts in a manner opposite to that of hexamethonium ion

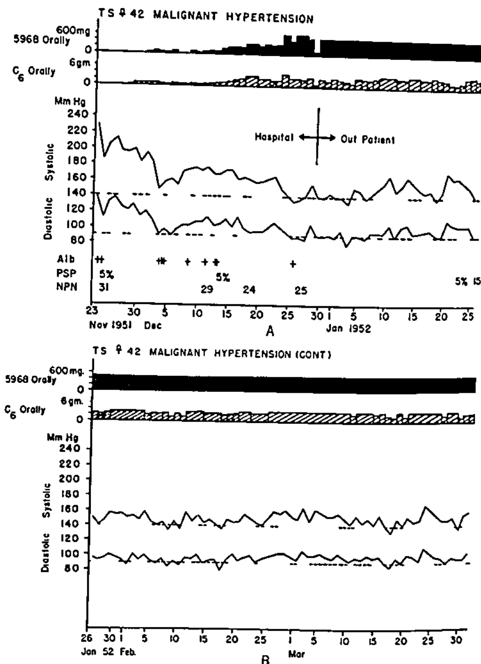
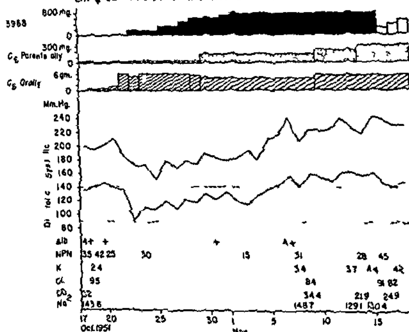


Fig 158 —Record of T S a forty two year-old woman with malignant hypertension and slight nitrogen retention on the basis of chronic pyelonephritis Ocular fundi were grade IV Blood pressure and the malignant stage were easily controlled She was re-admitted to hospital in May 1952 when it was found that her renal function had improved only slightly (5 per cent in fifteen minutes and 30 per cent in two hours) with a non protein nitrogen level in her blood of 30 mg per cent There was 1 plus albuminuria and positive cultures of the urine for colon bacillus In the succeeding months she had reached a therapeutic Grade of 1 Note the daily variations in dose of hexamethonium chloride $(C)_6$ Fundi shown in Color Plate III



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(Legend for Figure 1 3 on page 000)

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D B ♀ 56 HYPERTENSION SINCE 1944

A

D B ♀ 56 HYPERTENSION SINCE 1944 (CONT)

Hospital ← | → Out Patient

B

D B ♀ 56

C

FIG 159

Hepatic Function — Function of the liver as measured by the retention ... of the serum and cephalin-

the

ed

ture

mentation rate of erythrocytes ... blood cell count which may last a month or

p olongation of the P R interval ischemia ... other signs suggesting *left ventricular* strain In only 1 case of 252 did the electrocardiogram change adversely bundle branch block appeared ... appeared and reappeared at lower levels of blood pressure When the tracings suggested the presence of severe organic disease minor or no alteration in the normal direction occurred

... dilated hearts became smaller *when* the blood

quently accompanying the hypertensive state has been ... a number of instances Slow focal dysrhythmia caused by organic vascular disease has been unchanged by Hyphex

Non Protein Nitrogen in the Blood — In a number of cases normal NPN fell to rather low levels often as low as 10 mm per cent when Hyphex was given The explanation for this change is unclear

Pulsations in Peripheral Vessels — The peripheral arteries especially ... feel intensely constricted in severe hypertension one is

Pyruvic Acid Levels in Blood — Because of the strong binding of carbon group by 1 hydrazinophthalazine levels of pyruvic acid in blood were measured by Ferry No depression was found

EDEMA

Dependent edema can occur when Hyphex is begun and may even last for several weeks Generalized edema is not uncommon in women lasting a few days There are several types which can be distinguished clinically each from the other the best explanation for each is given although we cannot definitely prove the mechanisms

Generalized ...

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EFFECTS ON VARIOUS FUNCTIONS OF THE BODY

Naturally one is *curious to* discover whether or not continuous administration of potent drugs affects functions of the body *other than those* concerned with *their primary action* Therefore clinical laboratory procedures were performed on as many *criteria* as appeared reasonable Symptoms presented by *patients on Hyphex* no matter how trivial were translated into clinical tests *in order to make certain that obscure* disturbances were not present Likewise any *mechanism other than* cardiovascular which could readily be studied was *under suspicion* *No abnormalities were* found

Miscellaneous Studies

Basal Metabolic Rate —No depression or elevation *of the basal meta*bolic rate was observed in 100 patients which could be ascribed to the action *of Hyphex When the rate was elevated* without true hyperthyroidism *it often fell successively* in the hospital to slightly elevated levels possibly the *result of rest* The basal metabolic rate is normal in hypertension with the *exception of* approximately *20* per cent of those women exhibiting the *endocrine hypertensive* syndrome in whom it is elevated even in the *presence of obesity*

Carbohydrate Metabolism —Because 1-hydrazinophthalazine has an *affinity for metals* and the role of zinc in carbohydrate metabolism is *definite* studies were made to determine the effect of Hyphex upon fasting *blood* sugar and the tolerance of the level of blood sugar to injected or ingested glucose No disturbances were found A few patients experienced craving for sugar and sweets during the first three months of therapy *which disappeared* Tolerance to injected insulin has not been measured *but no reason for performing this test has arisen* Diabetes mellitus was uninfluenced

Body Weight and Nutrition —There was no loss of weight in patients taking Hyphex unless reducing diets were given *In fact some individuals* formerly obese and not on diets gained considerable weight two as much as 30 and one 40 pounds Obviously the drug did not interfere with nutrition nor was there any indication that they promoted excessive *accumulation of fat*

Adrenal Function —No clinical signs of hypo or hyperadrenalism have appeared While rough measurements of steroid excretion have not been performed alterations in the fasting eosinophile count in the peripheral blood have shown irregular changes which are not consistent in no case have eosinophils disappeared even during acute reactions

Plasma Cholesterol —Total levels of plasma cholesterol have decreased moderately in all cases both while the patients *were in hospital* and in the long term When elevated they became normal and when normal somewhat depressed This alteration has been consistent Its significance will take many years to evaluate Only in a few have levels reached values of 100 mg per cent

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hypertensive syndrome whose blood p
has responded to restriction of dietary salt, mercurial diuretics and other measures effective in promoting *diuresis*. When first discovered we were ... ttency which is quite charac-

COMMENT

of thinking are so firmly fixed that anything new ... their intellectual framework and from those who refuse to try the treatment for fear that it may be effective and thereby disturb the orderly progress of science. When a disease with such ... as hypertension is reversed or halted b... are made to explain results by such terms as ... for their lack of placebo controls

regimen so highly recommended by them ... a failure. The aim of therapy of hypert... do not obtain it in all cases enough patients change from severe hypertensives to mild ones to justify the belief that just a little more therapy directed at some other influence will produce normotension in all cases not ravaged by uremia or atherosclerosis (Table 103).

In the Hyphex method certain psychotherapeutic influences are undoubtedly at work. Usually they operate in reverse—when the drugs fail to maintain normal blood pressure the excitable patient becomes more excitable and his blood pressure rises further. There is certainly a confidence engendered by the fact that a patient can watch a pill lower his blood pressure; perhaps the emotional aspects of the disease are aided by that fact. We must insist, however, that these two drugs are effective and specific aside from any psychotherapeutic aspects, for they are effective in (a) dogs, (b) anesthetized rats, (c) human beings in coma, (d) anesthetized human beings, (e) human beings gasping for breath with pulmonary

quite obvious), tightness of rings on fingers, stiffness of fingers. The edema is very slight and disappears in a few days; it appears when the drug is first given or when a daily dose is raised. It resembles premenstrual edema. The best explanation is that histamine is not destroyed by the histaminase inhibited by the drug, the edema being caused by excess histamine. Antihistaminic agents are of some benefit in treatment but usually are unnecessary.

Dependent Edema Due to Hexamethonium Ion.—Because of the inhibition of vasoc ... methon ium ion ... nstrictor fibers ... ppeared requiring little treatment, although moderate dietary restriction of salt has been employed for short intervals. Relaxation of venous tone may be the only cause.

Dependent Edema Associated with the Endocrine Hypertensive Syndrome.—Severe edema has developed in 3 and moderate edema in 6 women exhibiting the endocrine hypertensive syndrome (see below). In 1 it appeared twice more than a year after Hyphex had controlled blood pressure at strictly normotensive levels. This edema is probably caused by primary renal retention of salt from excess salt retaining hormones which are apparently unaffected by Hyphex. In other words, the blood pressure can be controlled but the underlying adrenocortical disturbance remains.

RESULTS IN VARIOUS TYPES OF HYPERTENSION

Unfortunately for our diagnostic acumen, Hyphex is equally effective in neurogenic, nephrogenic and arteriosclerotic types. In cases exhibiting preponderant neurogenic influences, hexamethonium chloride is the drug with the greatest action, although no case has been controlled satisfactorily on it alone. In nephrogenic hypertension due to renal parenchymal disease both drugs are essential, although we have tended to depend upon somewhat larger doses of 1-hydrazinophthalazine. In nephrogenic hypertension due to atherosclerosis we have discovered that small doses of both drugs are adequate for control; the principal problem is over-control. The advent of Hyphex has made careful diagnosis of various types unimportant a ... icate treatment of accessory etiological ... Just as antibiotics have caused a lessen ... in infectious diseases, so Hyphex has resulted in less intellectual exercise in the exact diagnosis of hypertensive states.

Only in the endocrine hypertensive syndrome is it important to evaluate the type before applying therapeutic methods. About half of 20 cases were at first somewhat resistant to Hyphex. Most of the remainder exhibited signs from time to time which indicated that etiological factors were uninfluenced, although mechanism was controlled. It has become necessary to add low salt regimens to Hyphex in the resistant cases for a month or so in order to achieve adequate lowering of blood pressure, after which time dietary restriction was no longer necessary. All have since been well controlled.

the method in that condition for most of the individuals concerned were close to the end of their lives Similarly the high incidence of cerebral thromboses (with the total absence of cerebral hemorrhage) has merely ... merely dominated by

the method

The seriousness of the s... becomes manifest when e... wick* All patients in Stage ... those in Stage II 7 were in Group 4 13 in Group 3 and 2 in Group 2 The mortality rates in patients treated medically in one year according to Smithwick are ... Group 3 17 2 per cent Group 2 9 6 per cent ... they are Gr... cent The one year mortality rate due to hypertension ... treated by Hyphex is considerably lower than those surgically treated being 3 6 per cent total and 3 3 per cent excluding uremia

Obviously one must ask oneself whether it is not better to treat hyper... ... els is done rather than to wait until ... We do not know the answer All of ... we have encountered have been listed ... the human body accept without revul sion a constant intake of potent foreign chemical substances Only time will tell whether or not the hydrazines will cause new serious and even fatal diseases as they apparently did in the three Negro men who died (Table 97) Therefore the use of Hyphex represents a calculated risk it is indicated only when a physician s best judgment tells him that a patient will be in trouble within the next five years

Hyphex must be given as has been outlined Reason is the application of other men s experiences and mistakes to one s own problems The procedure as given helps to avoid a repetition of our own mistakes and promotes a repetition of our own successes To depart radically from that procedure invites serious consequences and what is worse an unreasonable accumulation of the same experiences which we have had to the detriment of the health of patients We sincerely hope that others will profit from our mistakes by avoiding them

We sincerely hope that Hyphex will be superseded by something better The method not only leaves much to be desired (frequent pill taking and initial side reactions) but does not lower blood pressure to strictly normal levels in individuals with cerebral vascular disease and severe atherosclerosis As the first effective method it represents only the beginning of a future bright with promise

Summary – A specific method for the control of arterial hypertension has been developed and used in 252 patients The results are far better than we have observed with any previously employed regimen or drug The method is only the first to be developed and leaves much to be desired One increment of the hypertensive process is probably not neutralized it

nor would there be in the mind of any physician who saw our patients that 63 individuals are now living who otherwise would have died. The results are little short of miraculous. Experience with them has pushed the method far beyond its limits which lie somewhere in the uremic state. Likewise the high death rate in congestive heart failure cannot deter us from use of

TABLE 103.—SUMMARY OF ALL PATIENTS ON HYPHEX 6 TO 24 MONTHS

	Total	Lost to follow up	Living and controlled therapeutic grade			Discontinued normotensive	Dead		Discontinued and hypertensive
			4	*3*	*2 1 0*		*On Hyphex*	*Discontinued*	
Malignant Hypertension	89								
Stage IVc	40			7	15		10	8	0
IVb	29	2		5	18		2	2	0
IVa	21	1 *		5	10		3	1	1
Benign Hypertension									
Stage III	139			26	96		2	4	11
II	23			0	18	2	0	1	2
Total	252	3		43†	157	2	17††	16‡	14

* Probably dead but discharged from hospital Grade 2

** Probably living but discharged from hospital Grade 1

† Grade 3 was chosen in 18 cases in order to avoid severe hypotensive reaction

†† Eleven died of diseases unrelated to hypertension

‡ All died of effects of hypertension

Chapter

22

DIRECTIONS FOR RESEARCH

[illegible] various facts

Thus [illegible]
and the [illegible]
state [illegible]
the most confused reader must admit that [illegible] have been raised rather than narrowed the field has opened for wider [res]earch and deeper probing into first causes the understanding of which [lea]d to therapy. The purpose of this last chapter is first to evaluate critically the various hypotheses advanced, point out their deficiencies [illegible] proven and warn of [illegible]

and direct searchings third to examine the [illegible] clinical and upon direct searching, upon clinical investigations and upon the national economy; fourth to describe as best we can the ideal therapeutic measures for control, cure and prevention; and fifth to discuss ways and means by which the ultimate goal can be attained. We are at a cross-roads and a wrong turning can set us back many years. For the short term goal, therapy has been lightly touched. The ultimate goals of cure and prevention however are nowhere in sight. Therefore this chapter is not only a *Zusammenfassung*, a pulling together, but a *Wegweiser*, a signpost for future probings.

CRITIQUE OF THE THEORIES

All physiological phenomena are eventually resolved in terms of physics and chemistry. All pathological phenomena can be similarly resolved in terms of physical or chemical changes. Knowledge of any one phenomenon is far from complete but the directions of researches must always be aimed toward that goal. In the following statements concerned with the theory of pathogenesis the goal often appears very distant and undiscernible.

Etiological Factors

Hypertension is a Psychosomatic Disease.—The basis for that statement is tenuous, the evidence indirect. All we really know is that certain members of the population react to emotional or painful stimuli by vasospasm, others presumably by viscerospasm or perhaps bronchospasm. The inherent chemical changes which cause these reactions are not understood.

lies somewhere in the realm of cerebral disorders When a better method is developed, Hyphex will become obsolete At present, however it is the best we have to offer sufferers from severe hypertension The problem of specific treatment no longer exists but the method of treatment has raised many new problems which must be solved

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separate and the metal is ready for refining. When that goal is reached Chapters 5, 6 and 7 can be condensed into a single paragraph.

PATHOGENETIC FACTORS

The Sympathetic Nervous System is Overactive.—The evidence for that statement is indirect; it is based upon both clinical and experimental observation and therapeutic agents known to act upon nerves. We must accept it however as a working hypothesis until proven otherwise. If so, is there absolute sympathetic overactivity or absolute parasympathetic underactivity? Is the deficiency hereditary or "catching"? Why is it overactive?

The answer lies in a more thorough understanding of the autonomic nervous system, especially of electrochemical reactions concerned in the conduction of nervous impulses and the biochemical reactions concerned in the formation of vasoactive amines and quaternary ammonium compounds. Our knowledge is limited. Perry and Gibbs chose one method for the synthesis of nor-epinephrine (from tyrosine, Chapters 10 and 14). Bever has chosen
in this process
better answers th
to react to stress by vasospasm lies somewhere in the realm of the chemical processes involved in the autonomic nervous system.

The Overactive Sympathetic Nervous System Eventually Causes Organic Renal Ischemia.—This statement is probably true when paragangliomata have been discharging into the circulating blood for several years. We have no proof however that organic renal vascular disease results from repetitive renal vasospasm. The statement is made on the most indirect evidence and has always constituted an intellectual stumbling block to the writer

ground. For reasons discussed below this statement is the most tenuous of the various links in the chain of the theory of pathogenesis. Upon due reflection one is forced to conclude that persons who develop hypertension have a specific renal (or vascular) deficiency which causes intermittent renal vasospasm slowly to become irreversible.

The Ischemic Kidney Releases Vasoactive Amines into the Circulation.—There is little doubt that this statement is true. Pherentasin is presumably of renal origin; for a long time we were reluctant to assign to it a non-phenolic configuration. Other amines are present in high concentrations. Ammonia in the urine is depressed. Proof of the statement is clear; the

explains it in part; there may be other substances however of similar activity which are not extracted from blood by our method.

Hypertension Causes Renal Arteriolar Disease.—Few will disagree at present. The experimental and clinical evidence is overwhelming. The

Hypertensive persons appear to be anxious, tense, and emotionally disturbed. Does this state represent the action upon cerebral function of trivalent nitrogenous compounds liberated as a metabolic fault by the hypertensive process or do anxiety and tension precede the onset of hypertension? Or do both processes work together, each implementing the other? We incline to this last explanation.

A study should be made on persons reacting to stress by vasospasm in order to discover whether or not nervous tension is present. It should be extended to include pre-hypertensive and mildly hypertensive persons and be controlled with persons not reacting by vasospasm. There are plenty of both types in the population. The answers may give further insight into the relations between psyche and soma.

Hypertension is a Disease of Western Civilization.—The basis for that statement is clear on the knowledge available but much ... is needed. First ... by studies condu... areas of the world ... of the same race exposed to different degrees of Western Civilization. India, for example, offers a variety of races and a variety of civilizations. Third, the various factors which make up what we call civilization must be examined: emotional stresses, food habits, exposure to petroleum, tar and smoke, contact with the products of the industrial revolution. The meaning of Western Civilization must be broken down into its component parts and each one carefully scrutinized for possible etiological significance in the production of vascular diseases. For we do believe that hypertension has been on the increase in the last 150 years and there must be some reason therefor which can be more carefully delineated than by the non-specific word "stress."

Hypertension is an Hereditary Disease.—All we know is that it runs in families. Do children "catch" it from their anxious vasospastic parents by learning to react as they do, or is the tendency of truly hereditary nature like baldness? Is the ability to react to stress by vasospasm confined only to persons exposed to Western Civilization, or is it a common trait in all men regardless of race and upbringing? Is that ability inherited? The answers can only be found by demographic exploration of other races than those in Europe and America.

—

pointed out. But the nature of that personality deserves further definition by critical appraisers. Is it inherited? Is it "catching," especially by children? Does it really precede hypertension? The answers may be suggested by the application of present techniques but not until psychology and psychiatry become sciences can we expect much advance in this area.

On an etiological basis, therefore, the areas are broad and ill-defined. Understanding will be slow, piecemeal, and will be expensive to attain. When the final answer is in, however, we can predict that it will be ridiculously simple. Man appears first to complicate and confuse simple processes in his feeble attempts at understanding, later the dross and slag

down the variables and consider less obstruse etiological factors than those of personality heredity and civilization Let us assume that the ut half the population of all In that event one or another ilized portions of the popula t the disease

t

contained in petroleum rubber aluminum ments intimately concerned with modern civilization should be examined for possible substances which could affect the evolution of hypertension There are two approaches therefore for the elucidation of etiology that involving psychosomatic pathways about which we know little and that involving extraneous substances about which we can know more without too much theorizing Let us then look at the possibilities in the second approach

POSSIBLE FUNDAMENTAL MECHANISMS

There are two fundamental disturbances which if understood would expose the two main roots of the problem Only by extensive biochemical study can they be explored One is concerned with the sympathetic nervous system the other with the kidneys or with enzymes concerned in the construction of smooth muscle Both are capable of solution

Defect in Autonomic Nervous System

The possible defects have been discussed in Chapters 8 and 19 By *deduction* we must assume an ability to react to stress by vaso pasm *through* the sympathetic nervous system To understand this process we must know first how is nor-epinephrine formed at sympathetic end organs second what are its chemical precursor third is there excessive production or insufficient inactivation? Demonstration of differences from normal in the metabolism of catecholic pressor amines in persons with hereditary predisposition to hypertension would provide a possible explana

hydroxyphenylalanine one might expect to find alterations in pigmentation of the skin If there were excess *production* of nor-epinephrine one might

one might
normal tha

explanation of how this happens is not at all clear Is it the result of over work of smooth muscle? Or of the "toxic" properties of pherentasin or of some other amine? Is it primarily physical or chemical? The answers will provide an important link in vascular knowledge

Renal Arteriolar Disease Causes Organic Renal Ischemia —There can be no disagreement by anyone We ourselves however have been struck by the apparent degree of reversible vasospasm in severe arterial hypertension We would be forced to believe on clinical grounds that renal vascular disease can sometimes be of relatively minor degree after many years of hypertension, but that vasospasm is very severe Otherwise Hyphex would cause uremia which has never occurred when kidneys were compensated

There … *-Pherentasin—Hypertension* … regoing It is strange that the … Goldblatt should be denied I … s in fact rather extraordinary for such a clear-cut demonstration of the experimental pathogenesis of a disease not to be applied to man by clinical investigators Reluctance to ascribe renal instigation in at least a proportion of cases can only be explained by emotional block or insufficient clinical experience The clinician who sees patients suffering from disease is often better fitted to think of pathogenetic and etiological influences than is the true 'scientist' who divorced from people applies statistics to prove or disprove clinical experiences The statistician might be able to prove the correlation between flat feet and dandruff the clinician knows that these two conditions are not in the present light of knowledge related Statistics are most misleading in physiology and pharmacology where the exceptional experiment may be more important than the rule in chemistry they are usually unnecessary for there is only one way for a reaction to go and variations are the result of poor control In medicine absolute control of experiments is impossible the variations in the test object man are innumerable and will never be understood

At present we believe that the burden of proof is on him who says that renal disease does not contribute to and influence hypertension There are too many experiments and too many clinical coincidences for any fair minded person to believe otherwise Insistence by clinically untrained investigators on the basis of statistics disgorged by a machine that this is not so is unconvincing We have been forced somewhat reluctantly it is true to accept the renal genesis of hypertension in some cases to reject it in others but mainly to believe that in few or no human beings except those with renal insufficiency does hypertension spring *sui generis* from the kidney without another factor operating The physician must accept all the facts as he finds them and draw his own conclusions to fit them the scientist can reject a certain minority of facts as uncontrolled unexplained statistically insignificant or unnecessary to his hypothesis of the majority His rejection however does not apply to the individual with whom the physician is concerned

Comment —The deficiencies in the theory of pathogenesis are obvious but the theory still remains the one of best fit When we follow the example of Nature who is fundamentally simple and lawful we can narrow

to that vitamin extensive clinical studies have yielded results of little or no significance ... of an enzyme system may have been broken by de... of the known ...

T ble 104 —In eraction B tween Met llic I ... A ti Nepi o Ent Ag nt

Metal	Con ma atoms per cent	968	BAL	SCN	I ease + D pressor −	Enzymes activated
Na	0 07	0	0	0	0	
K	0 06	0	0	0	−	
C	0	0	0	0	0	
Mg	0 01	0	0	0	−	Ca bo ypept dase ... tase ... rho ylase
F	0 006	+	0	+	−	C tylase ytoc ... lase per lase ... ogl ... ur ...ox dase
C	0 0001	+	0	Sl sol	+	Mo ph ... lase ... p dyphenol lase l crase ascorb ... x lase
Z	0 0002	0	0	0	0	Ins l ... rbo ... hyd ... peptidase ... ypept lase co-oxidase
Mn	0 00008	+	0	0	−	A g ... d ... ge ase ... lase ... pr lidase ... ypept lase ... pep lase
Co	0 00000	+	0			C ... ypept lase
V		+	+	+		T ... k ... ti ... flow ... rd tea
Se & T		+			−	... for ... base
Be		0				Competes with Mg ... phosphatase
Pb B ...		0				Reported ... on n ...
M & B				M		Esse ... lase
M		0	+			H v met l poise
B Sb Hg A			+	Hg		H vy ... l p ...
M						
				0		I foud ... I for ... k ...
Al		0				
C		0				
Ag		+		Sol 20γ[27]		

T ghtly bou d t ... te ... Inpl ed by m ... g ... 551
O idizes 998 (F ... + & V ... V(OH) ...
Decomposes H_2O (C ...
On ... jection

Metals Deficiency of an inorganic coenzyme could explain the renal defect quite adequately if we knew which metal and which enzyme was involved ... some me ... tensive ... shown the affinity of hydrazine derivatives BAL and thiocyanates for

the explanation lies in the sympathetic nerve ending and the chemical processes involved therein

Defect in Kidneys

Again by deduction the conclusion is drawn that the kidneys of persons reacting to stress by vasospasm are in some way deficient in recuperative powers so that transient ischemia leads to the production of pressor substances through insufficient deamination All of the known facts fit this conclusion the high levels of amines in blood altered amine patterns in urine, the low excretion of ammonia, the acid urine, the acid pH of the renal cortex and the depressed oxygen consumption There are three disturbances which come to mind to explain these findings (1) The kidney suffers from relative lack of oxygen (2) the kidney suffers from slow depletion of its deaminating enzymes (3) the kidney suffers from excess of oxidative enzyme inhibitors All of them or any one of them may be present

Oxygen Lack —In acute ischemic episodes deficiency of renal oxygen supply may set off vasospastic reactions On the other hand oxygen deficiency alone cannot explain what we see Such conditions as pulmonary emphysema congenital heart disease with anoxemia altitude sickness pneumonia right-sided congestive heart failure anemia Ayerza's disease cyanosis in general carbon monoxide poisoning in fact all diseases associated with deficient oxygen content in the blood do not necessarily cause hypertension most of them are in no way related Furthermore in nephrosis and acute nephritis renal oxygen consumption may be very low in the face of a normal plasma flow in heart failure it may be high in the face of a low flow While oxygen deficiency may produce vasospasm in some individuals it does not lead to hypertension in all Therefore on clinical grounds we must delegate the role of oxygen to second place realizing however the enormous oxygen consumption of the kidney and the fact that it is practically never normal in hypertension

Depletion of Deaminating Renal Enzymes —Depletion of those enzymes which form ammonia from amino acids may well explain the findings such as they are Here we are on most theoretical grounds but it is justifiable to consider how this can occur We have expended much time and thought in our laboratories upon the matter with very little return and yet understanding of the mechanism of nephrogenic hypertension lies here The deficiency may be in the protein of the enzyme in one or many organic coenzymes or in some inorganic coenzyme Let us consider each in detail

Specific protein deficiency is possible but unlikely Alteration of a protein molecule to inactivate its enzymatic activity is more likely but we understand very little of how this can happen other than by using destructive or traumatic methods

Deficiency of an organic coenzyme is a good possibility However most of the known coenzymes (vitamins) and many others (succinic pyruvic glutamic ascorbic acids, cytochrome C) of coenzyme activity have been given to animals and patients with chronic hypertension without effect Because of the transaminating action of pyridoxal we have been attracted

a powerful pressor substance * selenium which substitutes for sulphur and causes disturbances of the nails and hair tin nickel and aluminum Vana- [illegible] cent in malleable aluminum alloys in petroleum [illegible] rolonged

[illegible]
concerned in relaxation of smooth muscle

Selenium, by replacing sulphur in sulfhydryl system could inactivate oxidation reduction systems concerned in the destruction of pressor substances Selenium is widely used in rubber glass enamel it occurs in wheat grown in certain areas and poisoning has been described in man Theoretically curly hair would become straight and nails ridged if poisoning in man were related to the disease in animals It is depressor in brief experiments

Tin appears unlikely as an active agent although it is widely used [illegible] in canned acid foods and is absorbed into the [illegible]

[illegible] oked in [illegible] compleyes Little is known of its action except that it is [illegible] n intravenous injection

Aluminum is absorbed from food but has never been shown to be toxic in amounts ingested Widely used as an antacid it does not cause hypertension when given in very large quantities We cannot implicate this metal in our hypothesis although it could replace boron if there were such a system Many other heavy metals exist in the body possibly as extraneous substances For example copper and molybdenum are somehow interrelated for deficiency of the former can be induced by excess of the latter We do not know whether or not molybdenum is essential for health although it forms the coenzyme of xanthine oxidase

The above discussion represents an example of the kind of searching into basic mechanisms which may prove productive The public health implications would be enormous if it should The evidence however is based only upon deduction and the known actions of anti hypertensive compounds The effects of these substances however may lie in entirely different spheres

The extremely interesting side action of the hydrazines in inhibiting histaminase may provide a clue if we knew how that reaction proceeded If histaminase contained a metallic coenzyme we could explain the results If histaminase contained an organic coenzyme which combined with hydra[illegible]

[illegible]
acids and the precursors of pherentasin thereby inhibiting the formation

It is of considerable interest that only the pentavalent form of vanadium was present Trivalent vanadium salts exhibited no demonstrable action Hydrazines reduce pervanadate in vitro to the trivalent form

metals as well as the known vascular activities of the various metals. It is noteworthy that all three antihypertensive substances combine with certain metals and that BAL is pressor in normal persons. It is also of interest that thiocyanate inhibits amino acid oxidation and is a strong solvent for many metals, that methylene blue, a sulfur compound, is pressor in normal subjects but was depressor in the three hypertensive ones to whom we gave it, that thiosulphate is said to be depressor and that pyrophosphates lower blood pressure in hypertensive rats. The common denominator of all antinephrogenic substances known is an affinity for certain metals. The ten most obvious ones are discussed separatel... It must be remembered ...ent by another (inactive) ...fic therapeutic tools open ... the problem.

Essential Metals.—Copper is a coenzyme for tyrosinase, dopa oxidase, other polyphenol oxidases and ascorbic acid oxidase. Its presence is essential for the formation of indole and melanin from tyrosine. It is found in aluminum alloys, soil, plants and many other places. Mammalian copper deficiency is associated with depigmentation of the hair and skin. An uncharted field lies in the metabolic effects of the copper enzymes with opposing sulfhydryl complexes on the phenolic pressor substances and their inactivation. Why are Negroes more susceptible to hypertension than whites? In the skin, at least, there is a balance between the cupric coenzyme of phenolic (dopa) oxidase and sulfhydryl groups; ultraviolet light is said to inactivate the sulfur inhibitor, freeing the copper so that the enzyme will form pigment. We cannot, however, implicate excess of copper at present with any degree of suspicion, although it combines readily with hydrazines.

Iron and *zinc* do not fit into our hypothesis: anemia, carbonic anhydrase deficiency, carbohydrate disturbances and other profound metabolic disorders would result from the administration of the hydrazines. Both metals are essential for health, iron being concerned in many oxygen transport systems and zinc being an essential portion of insulin and carbonic anhydrase.

Manganese is a coenzyme for arginase, malic dehydrogenase, amino peptidase, dipeptidase and several other systems. It is essential for metabolism. Manganese occurs in cooking pots as alloys of aluminum, in the soil and in green plants. We cannot implicate manganese excess at present although it combines with the hydrazines.

Cobalt is a coenzyme for carboxypeptidase and is a constituent of vitamin B_{12}. It is a vasodilator of the skin and lowers blood pressure.[1] Cobalt fixes sulfhydryl groups within the tissues in stable combinations. Histidine and perhaps ascorbic acid also chelate with cobalt. Cobalt is necessary for the growth of certain bacteria in the gastrointestinal tract; whether or not these organisms synthesize other vitamins than B_{12} is unknown. Cobalt salts cause polycythemia in rats and rabbits. Deficiency in animals produces anemia and wasting.

Metals Replacing Essential Coenzymes.—It is possible that an extraneous metal may substitute for an essential one on the protein of the enzyme, inactivating enzymatic processes. Of these, the most likely are vanadium

n t t 1 a different

(

inactivated substance

If as Perry has demonstrated for urine sulfhydryl deficiency is a common accompaniment of hypertension we need to know whether this deficiency is primary to the nephrogenic chain of events or secondary Are SH group absent because they have chelated with metals? Interestingly enough adrenocorticotrophic hormone which can produce hypertension has been reported to lower the level of active SH groups in the blood It

nt t tory substances to vascular construction

they are in the kin in the tyro inase-DOPA

A highly theoretical schema is shown in Figure 161 in which all of these isolated but obviously related observations are portrayed We offer it

f 1 1+ t in any manner is a fin hed

REMOTE EFFECTS OF CONTROL

r 1 n 1 1 lh 1 control of their hyper

(b) that it will not produce other serious or fatal diseases which neither are not disproven As secondary physical and

estigators — Because con pharmacologists and clin ical investigators will spend a large amount of time money and effort in

uffer from it Improvement of chemotherapy is highly necessary But advance along this line in order to obtain a drug just a little better (or much better) than those now available must not be made at the expense of further understanding of pathogenesis There was no greater deterrent to advance in understanding of the pathogenesis of diabetes than the discovery of insulin The fascination of being able to control diabetes has distracted attention away from basic causes and mechanisms and focused efforts ural consequence of a physic without understanding laws This diversion of effort must not happen in the case of the hypertensive diseases

of amines, (
constriction (
involved in any one of the above systems * It is also possible that each anti nephrogenic substance could act on a different link in the chain

Depletion of Substances Inhibitory to Vasoconstriction —Still another possibility concerns the depletion from the body of substances which inhibit vasoconstriction thereby disturbing the delicate balance in smooth muscle of constriction and relaxation The important discovery by Perry *that the sulfhydryl content of hypertensive urine is markedly diminished* provides food for thought Sulfhydryl substances are anti hypertensive how they act is not known They are concerned in many reactions of

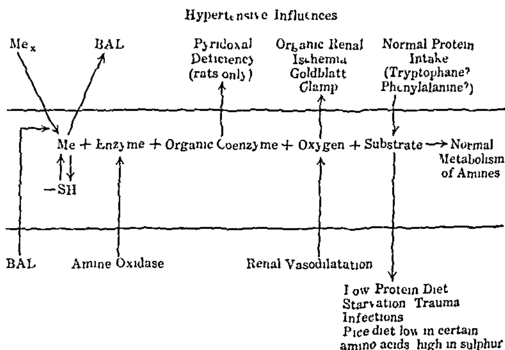


Fig 161 —Highly theoretical schema of possible fundamental mechanisms disturbed in nephrogenic fact
by an hypothesis
refers to a normal
the action of the normal one These influences apply only to man

oxidation reduction and in binding and releasing metallic coenzymes The pressor activity of BAL in normal persons is difficult to explain by any theory which assumes that flooding the body with sulfhydryl substances provides an excess of reducing agents However if a metallic or organic

* If (1) were true the direct action of the hydrazines on intra arterial injection would have to include either direct chemical inactivation of normally present vasoconstrictor subst
blood
not r
would contain increased quantities of a metal a most difficult measurement to make

Effect of Control on the Nation ... States is aging. Every year m... an has not been extended since ... as changed remarkably (Fig. 162) ... control and cure of infectious di... es, high standards of living and better preventive medicine have been ... the average age of the popula...

Assuming that every individual suffering ... (... H... assumption) approximately one-quarter of deaths ... about twice as ... In Figure 163 is shown the average lifetime expected for each age if both hypertension and atherosclerosis were controlled. The curve for hypertension lies a little less than half way between the 1939–1941 curve and the predicted curve

Age	Expectation of life 1939–1941	Years gained	Corrected expectation	Per cent increase
1	64 18	1 39†	66 37	2 13
10	03	1 45	58 48	2 54
20	4 6	1 45	49 21	3 03
30	38 80	1 45	40 25	3 73
40	30 03	1 43	31 46	4 6
50	21 96	1 34	23 30	6 10
60	15 05	1 11	16 16	7 3
0	9 42	0 6	10 18	8 06
80	3 06			

Above that expected in 39–41

† At age 0

(Calculated from Dublin, Lotka, Spiegelman: Length of Life. A Study of the Life Table. Revised Edition. New York: The Ronald Press Co., 1949; by Davies, D. F.: Mortality and Morbidity Statistics II. To be published.)

In Table 103 is given the impact on the population of control of cancer and in Table 104 that of control of cardiovascular and renal diseases. Half the white male population would live to the age of 88.6 years if the latter occurred. Obviously the economic and social problems would be enormous and a marked evolution in our attitude toward old age would be forced upon us.

IDEAL THERAPEUTIC METHODS

Immediate.—The short term goal, a practical method for controlling all cases of hypertension, has been reached. The method itself leaves much to be desired. The average patient takes 20 pills a day and takes his own

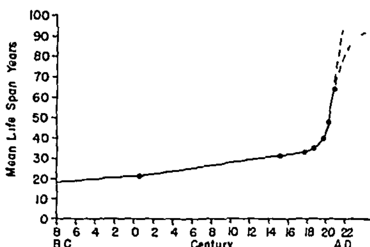


FIG 162 —Estimated change in mean life span The dotted lines indicate the two directions possible during the twentieth and twenty first century barring atomic war On clinical grounds the dotted curve to the right is the more logical one as mean life

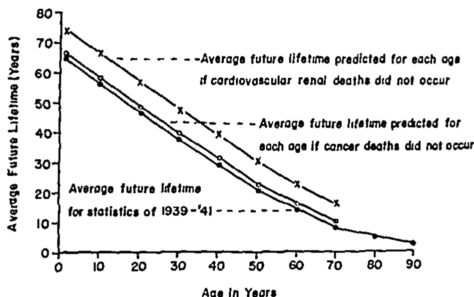


FIG 163 —Average future lifetime predicted on figures of 1939 1941 if deaths due to cancer and cardiovascular renal disease did not occur Actually the statistics for 1953 should show a shift to the right due to the influence of antibiotics the curve is therefore a conservative estimate (Chart prepared by Davies)

J ... specific drugs control and cure of infectious ...

is shown the average lifetime expected for each age if both hypertension and atherosclerosis were controlled. The curve for hypertension lies a little less than half way between the 1939-1941 curve and the predicted curve.

TABLE 105.—AVERAGE EXPECTATION OF LIFE AT EACH AGE (BY DECADES) IN WHITE MALE, ACCORDING TO LIFE TABLE FOR 1939-41 AND PREDICTED ON ASSUMPTION THAT DEATH DUE TO CANCER IS ELIMINATED

Age	Expectation of life 1939-41	Years gained	Corrected expectation	Per cent increase
1	64 98	1 39†	66 37	2 13
10	57 03	1 45	58 48	2 54
20	47 [illegible]	1 45	49 21	3 03
30	38 80	1 45	40 25	3 73
40	30 03	1 43	31 46	4 76
50	21 96	1 34	23 30	6 10
60	15 05	1 11	16 16	7 37
70	9 42	0 76	10 18	8 06
80	3 06			

Above that expected in 1939-41
† At age 0

(Calculated from Dublin, Lotka, Spiegelman: Length of Life: A Study of the Life Table. Revised Edition, New York, The Ronald Press Co., 1949, by Davies, D. F.: Mortality and Morbidity Statistics II. To be published.)

In Table 105 is given the impact on the population of control of cancer ... Half ... the latter ... enormous ... and a marked evolution in our attitude toward old age would be forced upon us.

IDEAL THERAPEUTIC METHODS

Immediate ...
cases of hyper...
be desired ... The average patient takes 20 pills a day and takes his own

blood pressure 5 times a day (he does not find this ordeal restricting because his symptoms are relieved and his blood pressure is lower) Control must be induced in hospital The primary action of hexamethonium ion on the parasympathetic nervous system causes distressing effects early in treatment which must be counteracted specifically

TABLE 106 —AVERAGE EXPECTATION OF LIFE AT EACH AGE (BY DECADES) IN WHITE MALES ACCORDING TO LIFE TABLE FOR 1939–41 AND PREDICTED ON ASSUMPTION THAT CARDIOVASCULAR RENAL DISEASES WERE ELIMINATED

Age	*Expectation of life 39–41*	*Years gained*	*Corrected expectation*	*Per cent increase**
1	64 98	9 01†	74 0	13 68
10	57 03	9 54	66 6	16 72
20	47 76	9 59	57 35	20 07
30	38 80	9 68	48 48	24 94
40	30 03	9 72	39 75	32 36
50	21 96	9 50	31 46	43 26
60	15 05	8 88	23 93	59 00
70	9 42	7 78	17 20	82 59
74	7 7	6 9	(14 6)	
80	5 38		±(10 00)	
90	3 06			

* Above that expected in 39–41
† At age 0

of (
age
or about 90 per cent more extra years than they would have expected at that age in 1940 This is based on the large assumption that persons likely to die of cardiovascular renal disease would have the same susceptibility to other diseases as occur in the general population As a group they might become either more or less susceptible to cancer for example than the rest of the population

(Calculated from Dublin Lotka Spiegelman Length of Life A Study of the Life Table Revised Edition New York The Ronald Press Co 1949 by Dyar D E Mortality and Morbidity Statistic II To be published)

An improvement of the method therefore requires the following in progressive order of possible attainment

1 Substitution for hexamethonium ion of a drug which acts specifically on the sympathetic division of the autonomic nervous system It must be taken orally (requirement of injections is backsliding) its effect last four to six hours and it must not cause tachycardia One cannot help but wonder whether or not duodenal ulcer would result from such a drug

2 Prolongation of action of this drug to last twenty four hours In that event a dose could be taken every morning or every evening and blood pressure would need be measured only once a day

3 Prolongation of action of a hydrazine derivative for the same period In that event, the recording of blood pressure by the patient could be reduced or eventually eliminated

4. A drug which slowly inhibits the sympathetic nervous system more or less permanently. In that event one agent eventually could be eliminated from the regimen.

5. A drug acting like the hydrazines which assuredly causes no late toxic effects or new diseases.

6. Specific anti-adrenal steroid compounds for treatment of alterations other than hypertension found in the endocrine hypertensive syndrome.

These are the ends in sight for the method of control. Each is possible in the relatively near future.

Distant.—Research into first causes and fundamental mechanisms concerned with the contraction and relaxation of smooth muscle can lead to therapeutic methods based upon those mechanisms. Many other methods for inhibition of neurogenic vasoconstriction can be found; many other methods for destruction of pherentasin and other vasoactive amines can

chemist directed by the clinician and the pharmacologist may be able to produce such a double-barreled weapon.

The distal goal, however, is cure of the disease, and cure can come only through understanding of pathogenesis. Cure is usually achieved by permanently breaking a cycle. The cycle cannot be broken logically until it is first understood. We do not believe that cure, at least in early stages of the disease, is too far distant for us to glimpse.

The serious and fatal end results of hypertension are often caused directly by atherosclerosis. Therefore the control of elevated blood pressure is only one major step towards prolonging life. The ability first to halt and later to reverse the vascular change of atherosclerosis by specific measures is an essential concomitant of treatment of hypertensive vascular disease in its wider aspects. Light on pathogenetic mechanisms is beginning to be shed but few practical measures have demonstrated therapeutic usefulness. [illegible] methods are developed, achievement of the goals for hypertension will leave the problem only half solved.

THE ULTIMATE GOAL

The ultimate goal of all clinical investigation is *prevention of disease*, and prevention can come only through understanding of *etiology*. Medical science has not achieved its end until it has learned to prevent as well as to control, treat or cure a disease. Treatment is only temporary, a stopgap to alleviate suffering *while medicine goes* on to prevent suffering of others fated to get the disease. When hypertension is well controlled by artificial methods, people will continue to develop it. But there will be a tendency to devote our energies and our funds to other pressing problems *or complacently*, and with some *justification*, to spend our time in routine treatment and minor improvements. Our interests must be kept alive and sufficient money to back our *interests be fully* available *after* all hypertension is controlled. The aim is complete understanding *leading* to prevention.

How can that goal be reached? By research, research, and more research, that is the only path. Research takes money, men and laboratories to urge knowledge onward. Money invested in research brings the largest dividends if the men who spend it are qualified. The public is now extremely "research conscious." The word itself, often has become unduly dignified by a halo of sanctity and spelled with a capital letter. But many organizations continue to lock the stable door after the horse has left. Service for the sick, care of the terminally diseased, periodic examination of the crippled, fruitless attempts to halt the advancing tides of Nature's processes when they have once begun, are expensive, continuing and ultimately unrewarding ways to spend money.

Research takes men and trained men. The wisest way to spend research money is to back men. But qualified men are scarce. Still scarcer are men with interests in the problems the public wants solved. Even more scarce are men with interest, intelligence, imagination and intuition who are able to spend most of their time on problems related to the health of the public. World War II set back the education of qualified investigators; we are still suffering from shortages in many fields.

Man, however qualified, has his limitations of learning. No one man can know enough to investigate a disease in all its biochemical, physical, physiological and clinical aspects. This point has received too little recognition. We can expect from the most qualified individuals results equal only to their limitations. As many investigators as possible must be backed by money for "project researches" in the hope that their combined efforts may lead to the accumulation of knowledge. This, however, is not enough. Small *groups* of men trained in different disciplines and devoted to the study of one disease also must be supported. By this second method, the interdisciplinary approach, the trained minds of physiologists, chemists, physicists and clinicians are brought to bear on both the fundamental and clinical aspects of a disease, the whole problem integrated and the front of knowledge advanced. If constituted properly, members of such a team feed each other and grow on the stimuli given each to the other. The biochemist can never observe what the clinician sees, nor vice versa. Let us recognize the limitations in the training of men and encourage both individual and team research; thus can we expect the fullest return from our investments. Interest in a problem is the most important prerequisite to solution and is something money cannot buy.

Methods of Research

The methods are simple, the solution difficult. There are three stages. An hypothesis consistent with the experimental facts is set up in order to point the way. Each interacting mechanism is tested, proven or disproven. The hypothesis is altered as each link in the chain is forged by experiment. Evolution continues from the complex to the simple, from clinical observations to chemical and physical ones. The investigator goes from the sickbed to the laboratory and back to the sickbed for nourishment of his ideas. Minor hypotheses are tested in animals and in man in parallel advances. Gradually the puzzle takes shape until suddenly it

wonded unless

clears
1

group on pherenta in which took seven years ...
carbonyl reagent in one year and to a method for control of hypertension in two. Shelf research the hiphy and testing of one substance after another taken down from the chemical helf must be discouraged for that form of earching is the most unrewarding of all
... the most difficult is the understanding of etiology

SUMMARY

This book is concerned with what the author has learned and with what he thinks about arteri

end results of a disease are of little importance in ... by specific measures. The simplest explanations consistent *with the facts* have been preferred to those more complex for the author affirms the philosophy that Nature is conservative simple and lawful that physiology rests upon the firm bases of physics and chemistry and that single fundamental physical or chemical disturbances can evoke widespread secondary pathological reactions which appear on the surface too complex for understanding.

A theory of pathogenesis consistent with the known facts was developed in 1938. With the advance of knowledge various minor modifications have become necessary but the main hypothesis has stood the test of time and of repeated attempts at disproof. With specific therapy based upon the theory its foundations have firmed and we must now accept it as more than theory. The etiology of hypertension however remains upon a much less factual foundation and it is toward solution of problems in this area that researche must be directed (Fig 164)

As this book has been written the mind of the author has been directed of necessity toward many issues which in the past have been passed over or neglected. Certain inconsistencies and contradictions have been disclosed by the very act of writing. The reader will note an evolutionary change from beginning to end in the discussion of actions and reactions mechanisms and processes which have tended from the complex to the more simple. Just as understanding progresses from the interdigitation and integration of the work of many so have mechanisms become more clarified as we progress from the theoretical to the practical. We hope that the process of clarification will proceed at a more rapid rate and that this

book has provided impetus and momentum It has failed in its purpose if "confusion has become worse confounded"

No attempt has been made to be orthodox—or unorthodox—in our own field Science is knowledge and truth knowledge and truth are neither orthodox nor unorthodox Only in the sense of the Bishop of Glou-

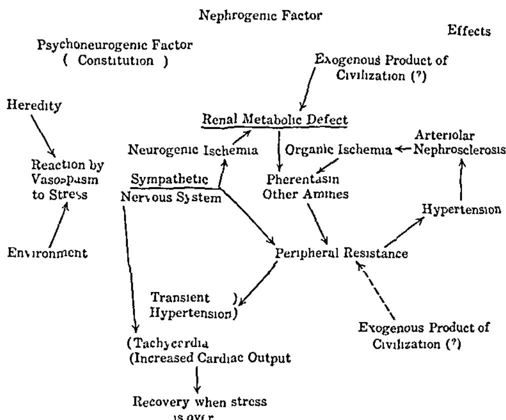


FIG 164 —Theoretical schema of sequence of events in pathogenesis and etiology of hypertension revised At the left are shown the factors concerned in the reaction to stress

in the oxidation of amines The familiar vicious circle is then begun which continues to progress as long as the neurogenic influence operates or when organic renal parenchymal disease or nephrosclerosis develops The metabolic defect however may exist in smooth muscle of arterioles rather than in the kidney An arrow omitted for simplification probably should be drawn from amine to brain indicating cerebral stimulation by these metabolic product

cester's 'Orthodoxy is my doxy heterodoxy is another man's doxy' can some of the theories expressed herein be considered a matter of one man's opinion Our opinions however are always subject to change when the light of new facts discloses their faults and inconsistencies We offer them for what they are rungs in the ladder leading to truth which will either bear the weight of tested facts or break through their inherent flaws

Nor have we att . . . . . d gmatic in fields other than our own particularly tho e c liam Scarlett sai l one becomes dogm conflict between Cl icine Pharmacolo Pathology and Physio g v the other The gaps in knowledge close but slowly the simplicity in genuity and regularity of fundamental dynamic processes are difficult to understand without faith that questions are answerable and problems solvable

Disease is the counterpart of health As health represents the beautiful economy of perfect function in disease is the ugliness of dysfunction The physician bears on his shoulders the enormous responsibility of attempting to restore perfect or adequate function with a very few specific tool The physician investigating disease has as his sole aim an understanding which can be utilized to control cure and prevent disease To gain that end he must discipline himself to discover disturbances in basic processes before attempting to treat disease this discipline is the most difficult part of the process of clinical research for physicians are trained to help human

hypertension can be controlled by a practical method that patients suffer

disease That is the ultimate goal of clinical research

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Appendix

A

THE PRESENCE OF A VASOCONSTRICTOR IN THE ARTERIAL PLASMA OF SOME INDIVIDUALS SUFFERING FROM ARTERIAL HYPERTENSION

THE following unpublished abstract was written in 1938 and represents our first attempts at detection of pherentasin. It is reproduced here for historical interest only, in that it stimulated purification and further exploration.

Dogs' tails freshly obtained were perfused with citrated plasma procured from the brachial artery and antecubital vein of individuals suffering from arterial hypertension. The blood was immediately centrifuged in the cold and the plasma stored in the frozen state. Perfusion pressure was kept constant at 70 mm Hg, temperature at 40° C, and the rate of perfusion measured on a smoked drum. In 7 individuals with arterial hypertension the arterial plasma markedly reduced the rate of perfusion, sometimes as much as 80 per cent, while the venous plasma showed no change as compared with normal venous plasma obtained from donors. In 3 patients with normal blood pressure and 1 normal dog the perfusion rates of arterial and venous plasmas were the same. In 3 patients with arterial hypertension this effect of arterial plasma (reduction of flow) was not obtained, nor was it in one suffering from coarctation of the aorta. Normal venous plasma obtained from five individuals gave no effect. One patient with uremia due to chronic glomerulonephritis had plasma, arterial and venous, which increased the perfusion rate. In one with very mild hypertension arterial plasma reduced the rate only slightly. Changes of less that 20 per cent were not considered significant. Viscosities of arterial and venous plasmas were approximately the same.

These results suggest the presence in the arterial blood of some hypertensive individuals of a substance which reduces the flow through a dog's tail and which is not present in venous blood.

Appendix

B

EXTRACTION OF PHERENTASIN FROM BLOOD

(Stock and Olsen's Method)

One volume of arterial blood is drawn in 3 volumes of cold 95 per cent alcohol. Add 95 per cent alcohol to total of 11 volumes (1 volume blood + 10 volumes alcohol). Stir gently and filter. To filtrate add 1 ml conc HCl for each 200 ml original blood. Evaporate almost to dryness under vacuo (nitrogen) and at temperatures not exceeding 35° C. To residue add 1 volume 90 per cent alcohol. Centrifuge and discard undissolved residue. Evaporate centrifugate under same conditions as above to aqueous slurry. Centrifuge and discard residue. Extract with washed petroleum ether and discard pet. ether washings. Add to aqueous solution made up to volume so that 1 ml is equivalent to 20 ml of original blood (Stock's Crude extract). 5 gm of Amberlite IR-100H for every 25 ml of extract. Allow to absorb with intermittent shaking for fifteen to thirty minutes. Decant off fluid and wash resin with ¼ volume of distilled water. Dilute resin with 1 volume of 5 per cent HCl overnight. Decant off aqueous layer and re-extract for one hour with ¼ volume of 5 per cent HCl. Decant. Mix aqueous layer and make alkaline and extract with $CHCl_3$. Shake intermittently for thirty minutes and separate off $CHCl_3$ layer. Evaporate $CHCl_3$ to dryness adding a drop of conc HCl. Take up residue in acidified water. Neutralize and assay (Olsen's Purified extract).

Properties of Olsen's Purified Extract

Blood extract pH circa 5.0

(1) Action of HCl

2 ml extract + 1 ml 0.1 N HCl—Δ boiling water bath two hours—cool—neutralize—assay—*positive*

(2) Action of NaOH

2 ml extract—neutralize* + 1 ml 0.1 N NaOH—Δ boiling water bath two hours—cool—neutralize*—assay—*negative*

(3) Action of HONO

2 ml extract + 0.7 ml 0.1 N KNO_2 + 1.3 ml 0.1 N HCl—allow to stand at room temperature for twenty four hours—with mild heating (warm water) and suction draw off excess HONO—neutralize*—assay—*negative*

(4) Action of NH_2OH
2 ml of extract—neutralized* + 1 ml 0 1 N NH_2OH HCl + 0 5
emperature
ositive (NH_2

(5) Action of Semicarbazide
2 ml of extract—neutralized* + 1 ml 0 1 N semicarbazide HCl + 0 75 ml 0 1 N NaOH—allow to stand at room temperature twenty four hours—neutralize—assay—*slightly positive* (semicarbazide in concentration used has no effect on assay)

(6) Action of Ketene
Extract in acid acetic anhydride buffer exposed to ketene (bubbling through) for ten minutes and for thirty minutes Solutions then made alkaline and extracted with chloroform Chloroform extract evaporated to dryness and taken up in acidified (HCl) water— neutralized* and assayed—ten minute fraction—*slightly positive*—30-minute fraction—*negative*

EXTRACTION OF PHERENTASIN FROM BLOOD
(Modified by Dean F Davies)

1 Bleed 1 volume (≅500 ml) of arterial blood directly into 1 500 ml of cold 95 per cent ethanol with agitation

2 Calculate final blood volume and dilute to 6 volumes with 95 per cent ethanol

3 Suction filter through Buchner funnel Rinse precipitate with two 50 ml portion of ethanol and suck dry

4 Add 1 0 ml concentrated HCl per 200 ml original blood volume Sample may be stored in cold room at this point

5 Vacuum distil to approximately $\frac{1}{10}$ original blood volume at < 35 C

6 Extract aqueous acid volume and precipitated solids with sufficient 95 per cent ethanol to make final concentration 90 per cent Centrifuge Rinse residue once with 50 ml 90 per cent ethanol Add rinse to extract Discard residue

7 Distil extract in vacuo at < 40 C to less than $\frac{1}{40}$ original blood volume

8 Acidify with 2 ml 0 15 N HCl and dilute to $\frac{1}{20}$ original volume with water

9 Transfer to 500 ml Erlenmeyer flask with petroleum ether and make to $\frac{1}{2}$ original blood volume with petroleum ether Shake for fifteen minutes on mechanical shaker (Be sure to use air vent cork)

10 Re-extract twice using $\frac{1}{10}$ original volume petroleum ether Discard ether extract which contains little or no vasoactivity

* Neutralized to pH 7 0

Appendix

B

EXTRACTION OF PHERENTASIN FROM BLOOD

(Stock and Olsen's Method)

One volume of arterial blood is drawn in 3 volumes of cold 95 per cent alcohol. Add 95 per cent alcohol to total of 11 volumes (1 volume blood + 10 volumes alcohol). Stir gently and filter. To filtrate add 1 ml conc. HCl for each 200 ml original blood. Evaporate almost to dryness under

C. To residue add

undissolved residue

to aqueous slurry.

Centrifuge and discard residue. Extract with washed petroleum ether and discard pet. ether washings. Add to aqueous solution made up to volume so that 1 ml is equivalent to 20 ml of original blood (Stock's Crude extract). 5 gm of Amberlite IR-100H for every 25 ml of extract. Allow to absorb with intermittent shaking for fifteen to thirty minutes. Decant off fluid and wash resin with ¼ volume of distilled water. Dilute resin with 1 volume of 5 per cent HCl overnight. Decant off aqueous layer and re-extract for one hour with ¼ volume of 5 per cent HCl. Decant. Mix aqueous layer and make alkaline and extract with $CHCl_3$. Shake intermittently for thirty minutes and separate off $CHCl_3$ layer. Evaporate $CHCl_3$ to dryness adding a drop of conc. HCl. Take up residue in acidified water. Neutralize and assay (Olsen's Purified extract).

Properties of Olsen's Purified Extract

Blood extract pH circa 5.0

(1) Action of HCl

2 ml extract + 1 ml 0.1 N HCl △ boiling water bath two hours—cool—neutralize—assay—*positive*

(2) Action of NaOH

2 ml extract neutralize* + 1 ml 0.1 N NaOH—△ boiling water bath two hours—cool—neutralize*—assay—*negative*

(3) Action of HONO

2 ml extract + 0.7 ml 0.1 N KNO_2 + 1.3 ml 0.1 N HCl—allow to stand at room temperature for twenty four hours—with mild heating (warm water) and suction draw off excess HONO—neutralize*—assay—*negative*

(4) Action of NH_2OH
2 ml of extract—neutralized* + 1 ml 0.1 N $NH_2OH \cdot HCl$ + 0.1
temperature
sitive (NH_2

(5) Action of Semicarbazide
2 ml of extract—neutralized* + 1 ml 0.1 N semicarbazide HCl + 0.75 ml 0.1 N NaOH—allow to stand at room temperature twenty four hours—neutralize—assay—*slightly positive* (semicarbazide in concentration used has no effect on assay)

(6) Action of Ketene
Extract in acid acetic anhydride buffer exposed to ketene (bubbling through) for ten minutes and for thirty minutes. Solutions then made alkaline and extracted with chloroform. Chloroform extract evaporated to dryness and taken up in acidified (HCl) water—neutralized* and assayed: ten-minute fraction—*slightly positive*; 30-minute fraction—*negative*

EXTRACTION OF PHERENTASIN FROM BLOOD
(Modified by Dean F. Davies)

1. Bleed 1 volume (≈ 300 ml) of arterial blood directly into 1,500 ml of cold 95 per cent ethanol with agitation.
2. Calculate final blood volume and dilute to 6 volumes with 95 per cent ethanol.
3. Suction filter through Buchner funnel. Rinse precipitate with two 50 ml portions of ethanol and suck dry.
4. Add 1.0 ml concentrated HCl per 20.0 ml original blood volume; sample may be stored in cold room at this point.
5. Vacuum distil to approximately $\frac{1}{10}$ original blood volume at < 35° C.
6. Extract aqueous acid volume and precipitated solid with sufficient 95 per cent ethanol to make final concentration 90 per cent. Centrifuge. Rinse residue once with 50 ml 90 per cent ethanol. Add rinse to extract. Discard residue.
7. Distil extracts in vacuo at < 40° C. to less than $\frac{1}{10}$ original blood volume.
8. Acidify with 2 ml 0.5 N HCl and dilute to $\frac{2}{10}$ original volume with water.
9. Transfer to 500 ml ... to $\frac{1}{2}$ original bloo... minutes on mech... aker. (Be sure to use ... vent cock.)
10. Re-extract twice using $\frac{1}{10}$ original volume petroleum ether. Discard ether extract which contain little or no activity.

*Neutralized to pH 7.0

11 Bring pH of aqueous layer after ether extraction to 5 0 with 1 N NaOH This extract corresponds to the "crude extract" of Stock and Schroeder

12 Add one-half weight per unit volume of neutral carboxylic-type cation exchange resin * Let stand in centrifuge tube with frequent agitation for ten minutes Centrifuge at high speed for fifteen minutes Decant and put filtrate aside Wash resin with equal volume of H_2O centrifuge, and discard waste water by decanting

13 Add 2 volu ... a isopropanol
and let s ... Centrifuge
Decant ... a isopropanol
Wash to eluate

14 Evaporate eluate to dryness in vacuum desiccator

15 Take up eluate in $\frac{1}{20}$ original blood volume of 95 per cent ethanol Centrifuge Decant Test residue for presence of amines

16 Evaporate to dryness in vacuum desiccator

17 Take up residue in $\frac{1}{10}$ volume of 0 15 N HCl in H_2O

18 Store in deep freeze for assay on rats before proceeding with chemical isolation

19 After demonstration of pressor activity the remainder of the extract is taken to dryness in a vacuum desiccator

20 The residue is taken up in 1 1 water isopropanol solution It is transferred quantitatively to a 3 to 5 cm strip of a paper chromatogram and run for eighteen to twenty four hours by capillary ascent in a solvent of 20 per cent water 30 per cent butanol and 50 per cent isopropanol

21 After drying a narrow vertical strip along the edge of the applied extract is cut to an inch beyond the solvent front separated from the rest of the paper and sprayed with Pyridine-Ninhydrin Reagent (see Appendix F on Estimation of Amines) The paper is warmed in front of an open drying oven at 105° C in a closed hood for one half hour The several spots which develop indicate the areas of the unsprayed portion to be eluted for further analysis of the amines present in blood These areas are eluted with 0 15 N HCl in water and used for assay electron diffraction and micro-analysis Pherentasin is the only known naturally occurring amine which has a pressor effect lasting at least ten minutes

* National Drug Company Cation Exchanger Carboxylic resin in powder form is prepared as follows Let recovered resin stand overnight with an excess of 0 1 N Na_2HPO_4 Rinse with water until wash is neutral Then let stand with M/15 phosphate buffer at pH 7 for 1 to 2 hours Filter and rinse resin with neutral phosphate buffer until wash is neutral Rinse excess buffer from resin with water until rinsings are sodium free

Appendix

C

BIO ASSAY OF PHERENTASIN

Rat Pressor Test.—Pherentasin concentrates as prepared under the preceding method may be assayed for pressor activity before further isolation is carried out. While the blood pressure response of rats to the [illegible] samples of pherentasin is marked and prolonged in [illegible]

[illegible]

on the same strain of rats. Some strains [illegible] intravenous drugs, others do not. The Wistar strain in our hands h[illegible]

[illegible]

cannulate with blu[illegible]

[illegible]

are satisfactory for snipping a V-shaped wedge in the artery. Inject [illegible] of 1 per cent heparin intravenously. Test animal for responsiveness to nor-epinephrine by injecting 1 [illegible] gamma per kilogram in the femoral vein. If the blood pressure rises less than 20/20 mm. Hg the rat should not be used for studies on pherentasin.

For assay, 0.[illegible] ml. of a sample made up in 0.15 N HCl and representing 20 ml. arterial blood is neutralized to pH 7.4 with solid sodium bicarbonate and injected intravenously into a renal hypertensive rat* of 200 grams or more. Systolic and diastolic pressures are recorded either by a Hamilton manometer, Sanborn electromanometer and recorder, or similar suitable

[illegible]

in extract of 20 ml. of blood which will, in at least 3 assays on 3 renal hypertensive rats, produce an average maximum elevation of diastolic blood pressure at ten, fifteen or twenty minutes after injection exceeding the 2 per cent level of probability for normotensive extracts. The blood pressure rise represented by a unit is therefore dependent on the strain of rats used and on the response of the rats to normotensive extracts prepared

* The left renal artery is partially constricted two or three weeks previously by tying a ligature about the artery and a thin wire 0.46 to 0.52 mm. in diameter, then removing the wire. Two-thirds of Wistar strain rats will develop hypertension when this has been done.

similarly The test is sensitive to approximately 0 2γ pherentasin based upon the weight of the purest sample

Rat Mesoappendix Test —The Chambers-Zweifach preparation is used to detect small amounts of pherentasin (approximately 0 02γ) The details of this test are described in "Methods for Medical Research," Vol 1 page 131 About 0 1 ml of the diluted extract is injected into the tail vein of a suitably prepared rat and the sensitivity of the vessels to the vaso constrictor action of adrenalin (Parke Davis) is observed The method is not specific for pherentasin

Bat Wing Test —The dermis of a section of a bat s wing is gently removed with a razor blade and covered with a glass cover slip, being kept moist with saline The bat is enclosed in a suitable rigid container attached to the platen of a microscope Pherentasin at pH 7 4 is applied directly to the surface under the glass and the intense constriction observed Generally it is necessary to wash the material off before the circulation returns to normal This test is sensitive but can only be done during the cold months when bats are collected from their places of hibernation

Appendix

D

KNOWN PROPERTIES OF PHERENTASIN

The following constitutes a résumé of all that is known of the nature and action of pherentasin

1 Small molecule non protein non fat Disappears upon dialysis in cellophane bag against running tap water overnight

2 Unstable Pressor activity lost when *crude* extract stand at room temperature overnight Must be kept frozen

3 Acid stable alkali unstable

4 Not inactivated by tyrosinase indicating absence of free phenolic group Inactivated by amine oxidase indicating presence of primary amine group

5 Soluble in water, 90 per cent ethanol chloroform alkaline toluene, insoluble in petroleum ether and acetone

6 Action destroyed by nitrous acid and ketene suggesting presence of amine group

7 Action destroyed or modified by hydroxylamine semicarbazide and 1 hydrazinophthalazine suggesting presence of carbonyl group

8 Has characteristic absorption spectrum with ninhydrin suggesting presence of amine group spectrum is dissimilar to that of any known compound Color formed is blue Peaks are at 410 mμ and 520 mμ

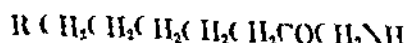
9 Has been purified to a substance giving two spots on paper chromatograms one with Rf value of 0 and one with Rf of 0 62 The latter contains the activity Normal blood extracts treated similarly give only spot with Rf of 0

10 Has x ray diffraction pattern unlike any known compound

11 Rf value suggests at least 5 carbon chain is present

12 Pressor activity in rat is slow to appear (two to three minutes) last at least fifteen to twenty minutes sometimes one to three hours

13 Formula of best fit at present



$$R\,CH_2CH_2CH_2CH_2CH_2COCH_2NH$$

Appendix

E

ESTIMATION OF AMINES IN URINE

(*Method of Davies and Perry*)

The following method is satisfactory for extraction, qualitative analysis and quantitative estimation of primary basic amines in urine. The catecholic amines are oxidized and the phenolic amines such as tyramine are poorly extracted.

Reagents

Carbon tetrachloride, reagent grade.

Ninhydrin reagent: 0.2 per cent ninhydrin in isopropanol.

Acid isopropanol mixture: Dilute 1 volume of concentrated hydrochloric acid to 100 volumes with isopropanol.

Water isopropanol: 1 volume of distilled water to 1 volume of isopropanol.

Pyridine, reagent grade.

Stock solutions of amine hydrochlorides: 0.01 M solutions of amines are made up in acid isopropanol mixture. Make all dilutions of stock solutions with acid isopropanol mixture.

Pyridine-Ninhydrin Reagent: Prepare 0.2 per cent ninhydrin in isopropanol containing 20 per cent pyridine.

Procedure.—Twenty four hour samples of urine collected under toluene are brought to pH 10.5 to 11.5 with sodium hydroxide, centrifuged, and the supernatant extracted for 12 hours with carbon tetrachloride in a Wolf-Hirshberg continuous extractor, the solvent being cycled through a flask containing 50 ml of 2.5 N HCl. The acid layer of the extract is extracted three times with equal volumes of petroleum ether and then concentrated to dryness in a vacuum desiccator with moderate warming. Ether layers are discarded. The dried residue is extracted with 2 ml water-isopropanol and transferred quantitatively to a filter using three 1 ml portions of water isopropanol as rinse. The filtrate is made up to 5 ml, mixed, and divided into two equal portions. One sample is applied quantitatively to a 5 inch strip on a sheet of No. 1 Whatman filter paper. To the second sample is added a mixture of 0.2 ml 0.01 M methylamine hydrochloride, *0.1 ml 0.01 M ethylamine hydrochloride* and 0.05 ml 0.01 M isoamylamine hydrochloride. This is applied on a second 5 inch strip and the mixture of the same quantity of known amines only is applied to a third strip. A hot plate on low heat can be placed under the paper to facilitate evaporation of the solvent from the paper. When the chromatogram is complete it is rolled into a cylinder according to the method of Williams and Kirby for ascending chromatography and placed

in a glass cylinder containing a solvent made up of 20 per cent distilled water 30 per cent butanol and 50 per cent isopropanol After running by capillary ascent for eighteen to twenty four hours the chromatogram is dried Each sample is divided into two fractions by cutting the paper up to the solvent front Half of each sample is sprayed with the pyridine-ninhydrin reagent The paper is warmed directly in front of an oven in a hood at 105 to 110° C for thirty minutes No amino acids are extracted by this procedure

The chromatogram is then examined for the presence of the known amines in the urine extract The Rf values of the known amines are modified by the presence of urine extract and also will be different in a mixture of amines than when chromatographed individually However inspection will tell whether the known amines are present as extra spots or as increased quantities of amines present in the undiluted extract If a spot occurs which does not coincide with one of the known amines an estimate of its chemical nature can be made by its Rf position and its color Table 107 indicates the ascending order of appearance of the amines

TABLE 107 —SELECTED PRIMARY AMINES BY ASCENDING Rf VALUES

2-Mercaptoethylamine	Nor Epinephrine
Glutamine	Ethylamine
Histamine	Tryptamine
Methylamine	2 Phenethylamine
Cystamine-2 HCl	Isoamylamine

which would be formed from the decarboxylation of some of their amino acid precursors The color of simple aliphatic amines under these conditions is blue to purple while that of ring substituted compounds is brownish blue to yellow The unsprayed portion of such a spot may be eluted and reapplied with a portion of known amine coinciding most closely with its characteristics Rarely do two separate amines have the same Rf value but final proof of identity requires methods not covered in this section

Elution of Amines and Their Ninhydrin Complexes – The spots and a ring of surrounding paper are cut from the chromatogram using precau

Each sample is cut

lenmeyer flask Two

volume depending on

are shaken on a shaking

The color in the filtrate

ometer at 405 and 570 mμ

wave length Absorption at 405 mμ is used as an estimate of quantity and the ratio of readings 405 to 570 give confirmatory evidence for the nature of the compound A simple aliphatic amine will have a 405/570 ratio below 1 20 the aryl-substituted and heterocyclic aliphatics have ratios above 1 30 Arylamines give no color with ninhydrin under these conditions By comparison of the absorption densities obtained from the undiluted extract with those of the mixture and with those of the known amines an estimate of the quantity of the amine excreted in the twenty four hour sample of urine can be made

Appendix

F

ESTIMATION OF HYDRAZINES IN URINE (PERRY'S METHOD)

A 100 ml aliquot is brought to pH 3 0 ± 0 2 and an ionic strength of about 0 5 exclusive of urinary electrolytes with glacial acetic acid and, when needed sodium acetate 0 50 ml of 2 4 per cent ninhydrin (triketohydrindine hydrate) is added we have found isopropanol more satisfactory as a ninhydrin solvent than water The mixture is heated to 70° C for seven minutes and then immediately cooled in an ice bath after which it is extracted by means of vigorous shaking for at least thirty seconds with 10 ml of chloroform When the two liquids have partially separated the chloroform layer which contains the yellow ninhydrin hydrazinophthalazine complex is cleared by centrifugation The color is stable for at least one hundred hours at 5° C The amount of hydrazinophthalazine in the urine is linearly proportional to the 460 mmu optical density of the chloroform solution is measured by the Beckman photoelectric spectrophotometer If the solution is too opaque to read it can be diluted with chloroform as much as necessary Under the conditions set forth above the following equation is a good approximation

Micromolarity of urinary C-5968 = 7 7 (460 mmu optical density − 0 10)

Appendix

G

DETERMINATION OF THE ASYSTOLIC ARTERIAL PRESSURE GRADIENT

THE asystolic gradient may be obtained in the forearm as follows Insert a needle connected to a Hamilton or other rapidly acting manometer into the brachial artery A pneumatic cuff is wrapped snugly about the upper arm and connected to a large reservoir of air under a pressure 50 mm Hg above systolic arterial pressure The connecting tubing is closed by a hemostat After recording fifteen seconds the hemostat is removed allowing the cuff to expand suddenly Recording is continued for twenty to thirty seconds or until the pressure ceases to fall Venous pressure does not rise under these conditions The curve is indicative of the degree of arterial and arteriolar constriction present in the arm The method may be modified to indicate patency of the Circle of Willis if the carotid has been exposed under anesthesia for small retrograde pulsations will appear in the record and the pressure will not fall below diastolic level when collateral channels from the opposite carotid artery are patent

Appendix

H

INTRADERMAL HISTAMINE TEST

The back

. . . forearm. A transient sharp pain similar to . . . for (1)

. . . the characteristic of previous type . . . (2)

. . . not . . .

. . . mistaken for a drug eruption. In dark Negro subjects . . . raphy is the only method known of detecting the reaction.

Appendix

I

DETERMINATION OF SODIUM AND CHLORIDE IN SWEAT

The subjects are seated in a room saturated with steam at a temperature of 110° F. The skin of the back is cleaned with alcohol. A canvas cover is hung above the patient in order to prevent precipitation of water on the back from condensation of steam on the ceiling. Fifteen and thirty minutes after exposure 1 or 2 ml. of sweat on the back are gently scraped off into test tubes with a flexible putty knife and analyzed for chloride and sodium. The chloride concentration follows that of sodium quite directly. Values below 20 mEq./L. in winter are considered significant; in very hot weather normal levels are half that of winter ones.

Appendix

J

LOEB'S FIVE RULES OF THERAPEUTICS

It is only fitting that this book end with a restatement of the five rules of therapeutics proposed by Dr Robert F Loeb in semi humorous vein many years ago. These rules apply to the treatment of hypertension as well as to that of other chronic and acute diseases.

Rule 1 The Golden Rule. Do not do anything to a patient that you would not like to have done to you.

Rule 2 If what you are doing is working effectively keep it up. Don't be a nervous therapist.

Rule 3 If what you are doing is not working stop it.

Rule 4 If you don't know what you are doing, don't do anything. Many iatrogenic diseases are caused by a compulsion on the part of the physician to do something with drugs now becoming increasingly more powerful.

Rule 5 Keep the patient out of the hands of the surgeon. This last rule applies, of course, to diseases caused by physical and chemical alterations which are in the main nonsurgical conditions. It does not, of course, apply in a surgical emergency or in diseases where removal of an offending organ or tumor is essential for life or health.

AUTHOR'S INDEX

SUBJECT INDEX

F

J

K

T

ERRATA

Page 328 Paragraph 2 Lines 9 and 10 Her plasma volume with a hematocrit of 43.5 per cent rose to 31.7 cc per kg and blood volume to 55.8 cc per kg Later the values were 37 and 74.7 cc per kg respectively without appreciable change in weight caused apparently by the addition of salt to her diet

Page 459 Fig 140 The formula of DOCA is incorrect See Fig 97, page 322

Page 385
Page 389 For dehydro when applied to compounds of ergot read dihydro
Page 475